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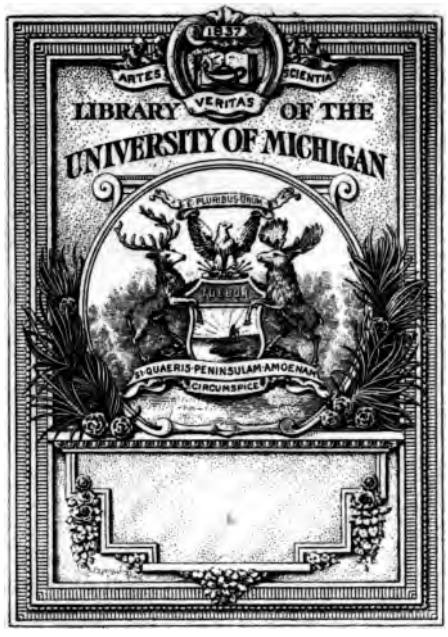
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VOLUME THE FIFTY-EIGHTH.

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1875.

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OCTOBER, 1875.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital; 13, Suffolk square, Cheltenham.

1851 *ACLAND, HENRY WENTWORTH, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Physician to the Radcliffe Infirmary; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

1842 †ACTON, WILLIAM, 17, Harley street, Cavendish square. *Trans.* 1.

1852 ADAMS, WILLIAM, Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 2.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, M.A. and M.D., F.L.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 38, Park square, Leeds. *Trans.* 3.

1863 ALTHAUS, JULIUS, M.D., Physician to the Infirmary for Epilepsy and Paralysis; 18, Bryanston street, Portman square. *Trans.* 2.

1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill, Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1870 ARNOTT, HENRY, Assistant-Surgeon to, and Lecturer on Morbid Anatomy at, St. Thomas's Hospital; 28, Brook street, Grosvenor square.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire, and 36, Sussex gardens, Hyde park. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Trans.* 8.

1817 †ASHBURNER, JOHN, M.D., F.L.S., 161A, Piccadilly, and 21, Hyde park place, Cumberland gate. C. 1821, 1830-31.

Elected

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

1836 BAIRD, ANDREW WOOD, M.D., Physician to the Dover Hospital; 7, Camden crescent, Dover, Kent.

1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital; 20A, Temple row, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 BAKER, WILLIAM MORRANT, Assistant Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. *Trans.* 2.

1869 BAKEWELL, ROBERT HALL, M.D., Dunedin, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General; Principal Medical Officer, Gibraltar. [6, Whitehall yard.] C. 1852-3. V.P. 1860-1. T. 1872. *Trans.* 2.

1848 BALLARD, EDWARD, M.D., *Vice-President*, Inspector, Medical Department, Local Government Board; 7, Compton terrace, Islington. C. 1872. V.P. 1875. *Trans.* 5.

1866 *BANKS, JOHN THOMAS, M.D., Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to the Coombe Hospital; 10, Merrion square east, Dublin.

1847 BARCLAY, ANDREW WHYTE, M.D., Physician to, and Lecturer on Medicine at, St. George's Hospital; Medical Officer of Health for Chelsea; 23A, Bruton street, Berkeley square. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. *Trans.* 2.

1862 BARKER, EDGAR, 21, Hyde park street.

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Trans.* 6.

Elected

1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 31, Grosvenor street. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Surgeon to the Charing Cross Hospital; 32, George street, Hanover square. *Trans.* 3.

1844 †BASHAM, WILLIAM RICHARD, M.D., Senior Physician to the Westminster Hospital; 17, Chester street, Belgrave square. S. 1852-4. C. 1860-1. V.P. 1864-5. T. 1871. *Trans.* 2.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.

1874 BAXTER, EVAN BUCHANAN, M.D., Assistant Physician to King's College Hospital; Professor of Materia Medica at King's College; 11, Weymouth street, Portland place.

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. *Trans.* 1.

1860 *BEALEY, ADAM, M.D., M.A. Camb., Oak Lea, Harrogate.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1836 BEAUMONT, WILLIAM RAWLINGS, Consulting Surgeon to the Toronto General Hospital; Toronto, Canada West. *Trans.* 3.

1871 BECK, MARCUS, M.S., Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square.

1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell. [Hanwell.]

Elected

1819 †BELL, THOMAS, F.R.S., F.L.S., The Wakes, Selborne, Hants. C. 1832-3. V.P. 1854. *Trans.* 1.

1871 BELLAMY, EDWARD, Senior Assistant-Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Professor of Anatomy in the Science and Art Department, South Kensington; 59, Margaret street, Cavendish square.

1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.

1845 BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.

1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1815 †BILLING, ARCHIBALD, M.D., F.R.S., Member of the Senate of the University of London; 34, Park lane. C. 1825. V.P. 1828-9.

1854 BIRD, PETER HINCKES, F.L.S., Medical Officer of Health for the Fylde Union, West Lancashire; 4, Clifton terrace, Lytham, Lancashire, and 1, Norfolk square, Sussex gardens, Hyde park.

1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6.

1851 BIRKETT, JOHN, F.L.S., *Treasurer*, 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-75. *Trans.* 8. *Sci. Com.*

1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.

1843 †BLACK, PATRICK, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 11, Queen Anne street, Cavendish square, C. 1856. V.P. 1866. T. 1869-70.

Elected

1840 BLAKISTON, PEYTON, M.D., F.R.S.

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street.

1867 BLOXAM, JOHN ASTLEY, Assistant-Surgeon to Charing Cross Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 BOJANUS, LOUIS HENRY, M.D., Wilna.

1846 BOSTOCK, JOHN ASHTON, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 54, Chester square, Belgravia. C. 1861-2. V.P. 1870-71. *Sci. Com.*

1869 BOURNE, WALTER, M.D. [care of the National Bank of India, 80, King William street, City.]

1870 *BOWLES, ROBERT LEAMON, M.D., 8, West terrace, Folkestone.

1841 †BOWMAN, WILLIAM, F.R.S., F.L.S., Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Trans.* 3.

1862 BRACE, WILLIAM HENRY, M.D., 7, Queen's Gate terrace, Kensington.

1874 BRADSHAW, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1867 *BRETT, ALFRED T., M.D., Watford, Herts.

1867 BRIDGEWATER, THOMAS, M.B. Lond., Harrow-on-the-Hill, Middlesex.

1868 BROADBENT, WILLIAM HENRY, M.D., Physician to, and Joint Lecturer on Medicine at, St. Mary's Hospital; Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Trans.* 1.

Elected

1851 BRODHURST, BERNARD EDWARD, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Trans.* 2. *Pro.* 1.

1872 BRODIE, GEORGE BERNARD, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 56, Curzon street, Mayfair. *Trans.* 1.

1844 †BROOKE, CHARLES, M.A., F.R.S., *Vice-President*, Consulting Surgeon to the Westminster Hospital; 16, Fitzroy square. C. 1855. L. 1866-72. V.P. 1875.

1857 *BROWN, ROBERT, late Surgeon to the Cumberland Infirmary, Wintershields, Brampton, Cumberland.

1860 BROWN-SEQUARD, CHARLES EDOUARD, M.D., F.R.S., Laureate of the Academy of Sciences of Paris; New York, United States. *Sci. Com.*

1874 BRUCE, JOHN MITCHELL, M.D., Assistant Physician to the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 8, Old Cavendish street.

1867 BRUNJES, MARTIN, 42, Brook street, Grosvenor square.

1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; Examiner in Materia Medica at the University of London; 23, Somerset street, Portman square.

1860 BRYANT, THOMAS, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Trans.* 8; *Pro.* 1. *Sci. Com.*

1855 BRYANT, WALTER JOHN, L.R.C.P. Edinb.; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., Inspector, Medical Department, Local Government Board; 24, Nottingham place Marylebone road.

1864 BUCKLE, FLEETWOOD, M.D.

Elected

1839 BUDD, GEORGE, M.D., F.R.S., Consulting Physician to the Seamen's Hospital, Greenwich; Ashleigh, Barnstaple. C. 1846-7. V.P. 1857. *Trans.* 5.

1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., F.R.S., President of the Royal College of Physicians; Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Trans.* 2.

1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Trans.* 4.

1873 BUTLIN, HENRY TRENTHAM, Surgical Registrar to St. Bartholomew's Hospital; Assistant Surgeon to the West London Hospital; 47, Queen Anne street, Cavendish square.

1871 BUTT, WILLIAM F., 12, South street, Park lane.

1818 BUTTER, JOHN, M.D., F.R.S., F.L.S., Physician Extraordinary to the Plymouth Royal Eye Infirmary; Windsor villa, Plymouth.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1861 CALLENDER, GEORGE WILLIAM, F.R.S., Surgeon to, and Joint-Lecturer on Surgery at, St. Bartholomew's Hospital; Lecturer on Anatomy and Physiology at the Royal College of Surgeons; Examiner in Anatomy at the University of London; 7, Queen Anne street, Cavendish square. C. 1874. *Trans.* 4. *Sci. Com.*

1874 CARR, WILLIAM, M.D., Lee Grove, Blackheath.

Elected

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 69, Wimpole street, Cavendish square, W. *Trans.* 1.

1845 CARTWRIGHT, SAMUEL, Professor of Dental Surgery at King's College, London; Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.*

1868 CAVAFY, JOHN, M.D., Assistant-Physician to, and Lecturer on Physiology at, St. George's Hospital; Physician to the Victoria Hospital for Children; 13, Arlington street, Piccadilly.

1871 CAYLEY, WILLIAM, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; Physician to the London Fever Hospital; 58, Welbeck street, Cavendish square.

1845 CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the Lock Hospital; 24, Mount street, Grosvenor square. C. 1861. V.P. 1867. L. 1869-72. *Trans.* 1.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1837 †CHAPMAN, HENRY THOMAS. C. 1858.

1868 CHEADLE, WALTER BUTLER, M.D., Assistant-Physician to, and Lecturer on Pathology at, St. Mary's Hospital; Assistant-Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.

Elected

1873 *CHISHOLM, EDWIN, Camden, near Sydney, New South Wales.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square.

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; Harley street, Cavendish square.

1860 CLARK, ANDREW, M.D., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square. C. 1875.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; 14, St. Thomas's street, Southwark, and The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Trans.* 5.

1848 CLARKE, JOHN, M.D., Physician to the General Lying-in Hospital; 42, Hertford street, May Fair. C. 1866.

1866 CLARKE, WILLIAM FAIRLIE, M.A. Oxon., Assistant-Surgeon to the Charing Cross Hospital; 12, Mansfield street, Cavendish square. *Trans.* 2.

1850 CLARKSON, JOSIAH, New Hall street, Birmingham. *Trans.* 1.

1842 †CLAYTON, OSCAR MOORE PASSEY, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

1853 CLOVER, JOSEPH THOMAS, 3, Cavendish place, Cavendish square. C. 1873.

1857 COATES, CHARLES, M.D., Physician to the Bath General and Royal United General Hospitals; 10, Circus, Bath.

Elected

1868 COCKLE, JOHN, M.D., F.L.S., Physician to the Royal Free Hospital; 7, Suffolk place, Pall mall. *Trans.* 2.

1850 COHEN, DANIEL WHITAKER, M.D., South Bank, North Down lane, Bideford, Devon.

1854 COLLINS, FREDERICK, M.D., Wanstead Lodge, Essex.

1865 COOPER, ALFRED, Surgeon to the Royal Hospital for Diseases of the Chest, City road, Additional Surgeon for Out-patients to the Lock Hospital; Assistant-Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.

1819 COOPER, GEORGE, Brentford, Middlesex.

1873 COOPER, GEORGE HENRY CRESSWELL, F.R.C.S. Ed.; Surgeon to the Holloway and North Islington Dispensary; Surgeon-Accoucheur to the Royal Maternity Charity; 35, Compton terrace, Highbury.

1843 †COOPER, WILLIAM WHITE, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.-P. 1873-4.

1868 CORNISH, WILLIAM ROBERT, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.

1860 *CORRY, THOMAS CHARLES STEUART, M.D., Surgeon to the Belfast General Dispensary; 146, Donegall Pass, Belfast.

1839 *CORSELLIS, CHARLES CÆSAR, M.D., F.L.S., Benson, Oxon.

1853 CORY, WILLIAM GILLETT, M.D., 47, Rue Tour Notre Dame, Boulogne-sur-Mer.

1847 †COTTON, RICHARD PAYNE, M.D., Consulting Physician to the Hospital for Consumption, Brompton; 33, Cavendish square. C. 1863.

Elected

1828 †COULSON, WILLIAM, F.L.S., Consulting Surgeon to St. Mary's Hospital, and to the German Hospital; 2, Frederick's place, Old Jewry, and 1, Chester terrace, Regent's park. C. 1831. L. 1832-7. V.P. 1851-2. *Trans.* 1.

1864 COULSON, WALTER JOHN, Surgeon to the Lock Hospital, 29, St. James's place.

1860 †COUPER, JOHN, Surgeon to, and Lecturer on Surgery at, the London Hospital; Assistant-Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street.

1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 19, George street, Hanover square.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Wiesbaden. C. 1853-4.

1868 CRAWFORD, THOMAS, M.D., Deputy Inspector-General of Hospitals (India); Umbalah, Punjaub.

1873 CREIGHTON, CHARLES, M.B., 2, Grosvenor villas, Wimbledon.

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1874 CRIPPS, WILLIAM HARRISON, 53A, Pall Mall.

1847 CRITCHETT, GEORGE, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Trans.* 1.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 61, Brook street, Grosvenor square.

1862 CROMPTON, SAMUEL, M.D., Physician to the Salford Royal Hospital and Dispensary; 24, St. Ann's square, Manchester.

1837 CROOKES, JOHN FARRAR, 5, Waterloo crescent, Dover.

1860 CROSS, RICHARD, M.D., Carlton House, Belmont road, Scarborough.

Elected

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

1846 CURLING, HENRY, Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 39, Grosvenor street. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Trans.* 13. *Pro.* 1. *Sci. Com.*

1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, London, and Assistant Physician to King's College Hospital; 3, Warwick street, Cockspur street.

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

1852 CUTLER, THOMAS, M.D., Spa, Belgium.

1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 1.

1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.

1848 DAUBENY, HENRY, M.D., San Remo, Italy.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 49, Rodney street, Liverpool.

1846 DAVIES, FREDERICK, M.D., Upton House, Ryde, Isle of Wight. C. 1873.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 18, Gay street, Bath.

1852 DAVIS, JOHN HALL, M.D., Physician Accoucheur to, and Lecturer on Midwifery at, the Middlesex Hospital; Physician to the Royal Maternity Charity, and Consulting Physician-Accoucheur to the St. Pancras Infirmary; Examiner in Obstetric Medicine at the University of London; 24, Harley street, Cavendish square. C. 1869-70.

Elected

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1867 DE MERIC, VICTOR, Surgeon to the Royal Free Hospital, and to the German Hospital, Dalston; 52, Brook street, Grosvenor square.

1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.

1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Clinical Medicine and Pathology at, St. George's Hospital; Physician to the Hospital for Sick Children; Examiner at the Royal College of Physicians, and at the University of Cambridge; 11, Chesterfield street, Mayfair. C. 1874-5. *Trans.* 12. *Sci. Com.*

1844 DICKSON, ROBERT, M.D., F.L.S., Cambridge Lodge, Harmondsworth, Slough. C. 1860.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting Ophthalmic Surgeon to the Asylum for Idiots; 29, Lower Seymour street, Portman square. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Trans.* 4.

1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; 84, Harley street. *Trans.* 2.

1845 DODD, JOHN.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to the London Hospital; 39, Welbeck street, Cavendish square. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1853 DRUITT, ROBERT, F.R.C.P. [8, Strathmore gardens, Kensington Mall.] *Trans.* 2.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 99, Southampton row, Russell square.

Elected

1865 DUCKWORTH, DYCE, M.D., Assistant-Physician to, and Lecturer on Skin Diseases at, St. Bartholomew's Hospital; 11, Grafton street, Bond street.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1874 DUFFIN, ALFRED BAYNARD, M.D., Physician to King's College Hospital; 18, Devonshire street, Portland place.

1874 DUKA, THEODORE, M.D., Surgeon-Major, H.M.'s Bengal Army; 38, Montagu square.

1871 DUKE, BENJAMIN, 1, Cavendish terrace, Clapham Common.

1871 *DUKES, CLEMENT, M.B. and B.S., Horton crescent, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1833 †DUNN, ROBERT, 31, Norfolk street, Strand. C. 1845-6. *Trans.* 2.

1861 DU PASQUIER, CLAUDIUS FRANCIS, Surgeon-Apothecary to H.M. the Queen, and to the Household of H.R.H. the Prince of Wales; 62, Pall Mall.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. *Trans.* 5. *Sci. Com.*

1874 DURHAM, FREDERIC, M.B., Surgical Registrar to Guy's Hospital; 38, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1839 DYER, HENRY SUMNER, M.D., Sennowe Hall, Guist, Norfolk. C. 1854-5.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

1836 EARLE, JAMES WILLIAM, late of Norwich.

1868 EASTES, GEORGE, M.B., Lond. Surgeon-Accoucheur to the Western General Dispensary; 5, Albion place, Hyde park square.

1824 EDWARDS, GEORGE.

1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.

Elected

1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.

1861 *ELLIOT, ROBERT, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle; Coroner for Carlisle; 35, Lowther street, Carlisle.

1848 ELLIS, GEORGE VINER, Professor of Anatomy in University College, London. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., Belle Grove Villa, Welling.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1842 †ERICHSEN, JOHN ERIC, late Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; 6, Cavendish place, Cavendish square. C. 1855-6. V.P. 1868. *Trans.* 2.

1874 EVANS, GEORGE HENRY, M.D., Assistant Physician to the Middlesex Hospital; 29, Devonshire street, Portland place.

1845 EVANS, WILLIAM JULIAN, M.D., Pinner, Middlesex.

1864 FAGGE, CHARLES HILTON, M.D., Assistant-Physician to, and Lecturer on Pathology at, Guy's Hospital; and Physician to the Evelina Hospital for Sick Children; 11, St. Thomas's street, Southwark. *Trans.* 4.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 8, Wood street, Doncaster.

1858 FALCONER, RANDLE WILBRAHAM, M.D., Physician to the Bath United Hospital; 22, Bennett street, Bath.

1862 FARQUHARSON, ROBERT, M.D., Lecturer on Materia Medica at St. Mary's Hospital; Physician to the Belgrave Hospital for Children; 23, Brook street, Grosvenor square.

1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 12, Hertford street, Mayfair. C. 1857. V.P. 1864. *Sci. Com.*

Elected

1872 FAYRER, JOSEPH, C.S.I., M.D., F.R.S. Edin., Honorary Physician to H.M. the Queen, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-Major, Bengal Army; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 16, Granville place, Portman square.

1872 FENWICK, JOHN C. J., M.D., 30, Devonshire street, Portland place.

1863 FENWICK, SAMUEL, M.D., Assistant-Physician to, and Lecturer on Medicine at, the London Hospital; 29, Harley street, Cavendish square. *Trans.* 3.

1841 †FERGUSSON, SIR WILLIAM, Bart., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon to King's College Hospital; 16, George street, Hanover square. C. 1849-50. V.P. 1863-4. *Trans.* 4.

1852 *FIELD, ALFRED GEORGE, Alverton Manor House, Stratford-on-Avon.

1849 FINCHAM, GEORGE TUPMAN, M.D., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

1866 FISH, JOHN CROCKETT, B.A., M.B. Camb., Junior Physician to the West London Hospital; 92, Wimpole street, Cavendish square.

1836 †FISHER, SIR JOHN WILLIAM, 33, Park Lane. C. 1843-4.

1860 FITZGERALD, THOMAS GEORGE, Surgeon-Major. [6, Whitehall yard.]

1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 7, Waterloo street, Birmingham. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1848 †FORBES, JOHN GREGORY, 82, Oxford Terrace, Hyde Park. C. 1868-9. *Trans.* 3.

Elected

1852 †FORSTER, JOHN COOPER, *Secretary*, Surgeon to Guy's Hospital; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. *Pro.* 1.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.

1859 FOX, EDWARD LONG, M.D., Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church House, Clifton, Gloucestershire.

1858 FOX, WILSON, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; Examiner in Medicine at the University of London; 67, Grosvenor street. C. 1875. *Trans.* 3.

1871 FRANK, PHILIP, M.D., Cannes, France.

1843 FRASER, PATRICK, M.D. C. 1866.

1868 FREEMAN, WILLIAM HENRY, 29, Spring gardens.

1836 †FRENCH, JOHN GEORGE, late Surgeon to the St. James's Infirmary; 10, Cunningham place, Maida hill. C. 1852-3.

1864 *GAIRDNER, WILLIAM TENNANT, M.D., Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.

1874 GALABIN, ALFRED LEWIS, M.A., M.D., Assistant Obstetric Physician to Guy's Hospital; Assistant-Physician to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Trans.* 1.

1865 GANT, FREDERICK JAMES, Surgeon to the Royal Free Hospital, 16, Connaught square, Hyde park. *Trans.* 2.

1867 GARLAND, EDWARD CHARLES, L.R.C.P. Edin., Yeovil, Somerset.

Elected

1867 GARLIKE, THOMAS W., Highfield, 126, Tulse hill, Brixton.

1854 GARROD, ALFRED BARING, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. *Trans.* 8.

1857 GASCOYEN, GEORGE GREEN, Surgeon to the Lock Hospital; Assistant-Surgeon to, and Joint Lecturer on Surgery at, St. Mary's Hospital; 48, Queen Anne street, Cavendish square. S. 1866-9. C. 1871-2. *Trans.* 4. *Sci. Com.* 2.

1851 GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875.

1819 GAULTER, HENRY.

1848 GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 10, Finsbury place south. C. 1874-5.

1866 GEE, SAMUEL JONES, M.D., Assistant-Physician to St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Harley street, Cavendish square. *Trans.* 1.

1821 *GEORGE, RICHARD FRANCIS, 20, Marlborough buildings, Bath.

1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; Physician to the Samaritan Free Hospital; 8, Upper Brook street, Grosvenor square.

1867 GOODEVE, EDWARD, M.B., Hon. Physician to H.M. the Queen; late Surgeon-Major, H.M.'s Bengal Army; Drimagh, Stoke Bishop, near Bristol.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Trans.* 2.

1873 GOWERS, WILLIAM RICHARD, M.D., Assistant-Physician to University College Hospital; 50, Queen Anne street. *Trans.* 1.

1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury Square.

Elected

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Heathfield, Ringwood, Hants. C. 1863.

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square.

1843 †GREENHALGH, ROBERT, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Bartholomew's Hospital; Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital; 72, Grosvenor street. C. 1871-2.

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Physician to, and Lecturer on the Practice of Medicine at, the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; 14A, Manchester square. *Trans.* 3.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Assistant-Physician to the Victoria Hospital for Children; 6, Curzon street, Mayfair.

1814 GROVE, JOHN, M.D., Salisbury.

1852 GROVE, JOHN, Spring Grove, Hampton, Middlesex.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris.

1849 GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Trans.* 4.

1837 GULLY, JAMES MANBY, M.D., Great Malvern, Worcestershire.

1854 HABERSHON, SAMUEL OSBORNE, M.D., Physician to, and Lecturer on Medicine at, Guy's Hospital; 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. *Trans.* 3.

Elected

1849 HAILEY, HAMMETT, F.L.S., Tickford Lodge, Newport Pagnell, Bucks.

1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.

1838 †HANCOCK, HENRY, Consulting Surgeon to the Charing Cross Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; 76, Harley street, Cavendish square. C. 1851. V.P. 1869.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; 13, Sussex place, Onslow gardens.

1836 HARDING, JOHN FOSSE, Ulverstone House, Uckfield, Sussex. C. 1858-9.

1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 57, Brook street, Grosvenor square. C. 1873-4.

1857 HARLEY, GEORGE, M.D., F.R.S., 25, Harley street, Cavendish square. C. 1871-2. *Trans.* 1. *Sci. Com.* 2.

1864 HARLEY, JOHN, M.D., F.L.S., *Secretary*, Assistant-Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 78, Upper Berkeley street, Portman square. S. 1875. *Trans.* 6.

1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.

1859 HARRIS, FRANCIS, M.D., F.L.S., 24, Cavendish square.

1872 HARRIS, WILLIAM H., M.D., Professor of Midwifery and Diseases of Women and Children, Madras Medical College, Madras.

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool.

1841 †HARVEY, WILLIAM, Surgeon to the Royal Dispensary for Diseases of the Ear and to the Freemasons' Female Charity; Aural Surgeon to the Great Northern Hospital; 2, Soho square. C. 1854.

1854 HAVILAND, ALFRED, Medical Officer of Health for the combined Districts of Northamptonshire; Northampton.

Elected

1870 HAWARD, J. WARRINGTON, Assistant-Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 5, Montagu street, Portman square. *Trans.* 1.

1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Trans.* 12.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Trans.* 2.

1848 HAWKSLEY, THOMAS, M.D., Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 6, Brook street, Grosvenor square.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square.

1861 HAYWARD, WILLIAM HENRY, Church House, Oldbury, Worcestershire.

1848 *HEALE, JAMES NEWTON, M.D., Medecroft, Winchester, Hants.

1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1874 *HEATON, JOHN DEAKIN, M.D., Senior Physician to the Leeds General Infirmary, and Lecturer on Medicine at the Leeds School of Medicine; Claremont, Leeds.

1829 †HEBERDEN, THOMAS, M.D., 98, Park street, Grosvenor square.

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1843 HEWETT, PRESCOTT GARDNER, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the Academy of Medicine, Paris; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Trans.* 7. *Sci. Com.*

Elected

1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square.

1872 HEYN, JULIUS CHARLES WILLIAM, M.D., 88, Lange Voorhout, the Hague, Holland.

1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to Guy's Hospital; 38, Brook street, Grosvenor square.

1862 HILL, M. BERKELEY, M.B. Lond., Surgeon to University College Hospital, and Lecturer on Operative Surgery at University College, London; Surgeon for Out-patients to the Lock Hospital; 55, Wimpole street, Cavendish square.

1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.

1841 †HILTON, JOHN, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Consulting Surgeon to Guy's Hospital; Consulting Surgeon to the Royal General Dispensary, St. Pancras; 10, New Broad street, City. C. 1851. V.P. 1863-4. *Trans.* 4.

1859 HIRD, FRANCIS, Surgeon to the Charing Cross Hospital; 13, Old Burlington street.

1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1872 HOGG, FRANCIS ROBERTS, M.D., India.

1843 †HOLDEN, LUTHER, Surgeon to St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; 65, Gower street, Bedford square. C. 1859. L. 1865. V.P. 1874.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., 7, St. John's terrace, Brighton.

1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road, South Hampstead.

1856 HOLMES, TIMOTHY, M.A. Camb., *Librarian*, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; Surgeon in Chief to the Metropolitan Police Force; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-5. *Trans.* 5. *Sci. Com.*

Elected

1846 HOLT, BARNARD WIGHT, Consulting Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3.

1846 HOLTHOUSE, CARSTEN, Surgeon to, and Lecturer on Clinical Surgery at, the Westminster Hospital; 3, George street, Hanover square. C. 1863.

1865 HOWARD, BENJAMIN, M.D., Lecturer on Operative Surgery, and Surgeon to the Long Island College Hospital, New York; 134, West 34th street, New York.

1865 HOWARD, EDWARD, M.D., Oaklands, Penge, Surrey.

1874 HOWSE, HENRY GREENWAY, M.S. Lond., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children.

1857 HULKE, JOHN WHITAKER, F.R.S., Surgeon to, and Joint Lecturer on Surgery at, the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. *Trans.* 4. *Sci. Com.*

1857 HULME, EDWARD CHARLES, Woodbridge road, Guildford. *Trans.* 1.

1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 5.

1866 HUNTER, CHARLES, 30, Wilton place, Belgrave square.

1873 HUNTER, WILLIAM GUYER, M.D., Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-Major, Bombay Army, Bombay.

1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

Elected

1856 HUTCHINSON, JONATHAN, Surgeon to the London Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields, and to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. *Trans.* 5. *Pro.* 2.

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., Senior Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.

1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., 1, Albert mansions, Victoria street, Pimlico.

1871 JACKSON, J. HUGHLINGS, M.D., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

1841 †JACKSON, PAUL, 24, Wimpole street, Cavendish square. C. 1862.

1868 JACKSON, THOMAS CARR, Surgeon to the Great Northern Hospital, and Surgeon to the National Orthopædic Hospital; 91, Harley street, Cavendish square.

1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington st., Wolverhampton.

1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.

1825 JAMES, JOHN B., M.D.

1839 JEFFREYS, JULIUS, F.R.S., 9, Park villas west, Queen's road, Richmond, Surrey.

1840 *JENKS, GEORGE SAMUEL, M.D., 18, Circus, Bath.

1851 JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., F.R.S., *Vice-President*, Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Trans.* 3.

Elected

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Genevois."

1847 JOHNSON, GEORGE, M.D., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-4. V.P. 1870. *Trans.* 10.

1868 JOHNSTON, WILLIAM, M.D., 44, Princes square, Hyde park.

1848 JOHNSTONE [JOHNSON], ATHOL ARCHIBALD WOOD, Surgeon to the Brighton Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Trans.* 1.

1862 JONES, CHARLES HANDFIELD, M.B., F.R.S., Physician to, and Lecturer on Clinical Medicine at, St. Mary's Hospital; 49, Green street, Grosvenor square.

1837 †JONES, THOMAS WILLIAM, M.D., 55, St. John's park, Upper Holloway. C. 1858.

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1872 KELLY, CHARLES, M.D., Medical Officer of Health for the West Sussex Combined Sanitary District, Horsham, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

1847 KEYSER, ALFRED, King's Hill, Berkhampstead.

1857 KIALLMARK, HENRY WALTER, 66, Princes square, Bayswater.

1851 KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Trans.* 1. *Sci. Com.*

Elected

1855 LANE, JAMES ROBERT, Surgeon to, and Lecturer on Surgery at, St. Mary's Hospital; Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Trans.* 1.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; Consulting Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865.

1865 LANGTON, JOHN, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square.

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 95 *bis*, Rue de Passy, Passy, Paris.

1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1816 LAWRENCE, G. E.

1840 LAYCOCK, THOMAS, M.D., F.R.S.E., Physician-in-Ordinary to H.M. the Queen in Scotland, Professor of the Practice of Medicine and of Clinical Medicine, and Lecturer on Psychology and Mental Diseases in the University of Edinburgh; 13, Walker street, Edinburgh.

1843 *LEACH, JESSE, Moss Hall, Heywood, Lancashire.

1868 LEARED, ARTHUR, M.D., Senior Physician to the Great Northern Hospital; 12, Old Burlington street.

1843 †LEE, HENRY, Surgeon to, and Lecturer on Clinical Surgery at, St. George's Hospital; Professor of Pathology and Surgery at the Royal College of Surgeons; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Trans.* 13. *Pro.* 2. *Sci. Com.*

Elected

1822 †LEE, ROBERT, M.D., F.R.S., Corresponding Member of the Academy of Medicine, Paris; 15, The Avenue, Berrylands, Surbiton, and 28, Maddox street, Bond street. C. 1829, 1834. S. 1830-3. V.P. 1835. *Trans.* 27.

1869 LEGG, JOHN WICKHAM, M.D., Physician to Casualty Department and Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; 47, Green street, Park lane. *Trans.* 1.

1836 LEIGHTON, FREDERICK, M.D., Frankfort-on-the-Maine.

1872 LIEBREICH, RICHARD, Ophthalmic Surgeon and Lecturer on Ophthalmic Surgery at St. Thomas's Hospital; 16, Albemarle street, Piccadilly.

1806 LIND, JOHN, M.D.

1872 *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

1852 LUKE, JAMES, F.R.S., Consulting Surgeon to the London Hospital; Woolley Lodge, Maidenhead Thicket, Berks. C. 1858. *Trans.* 4.

1857 LYON, FELIX WILLIAM, M.D., 4, Graham street, Edinburgh.

1867 MABERLY, GEORGE FREDERICK, Leamington, Warwickshire.

Elected

1873 MacCarthy, Jeremiah, M.A., Assistant-Surgeon to, and Lecturer on Physiology at, the London Hospital; 26, Finsbury square.

1867 Mac Cormac, William, M.A., Surgeon to, and Joint Lecturer on Surgery at, St. Thomas's Hospital; 13, Harley street. *Trans.* 1.

1862 *M'Donnell, Robert, M.D., F.R.S., Surgeon to Steevens' Hospital; 14, Lower Pembroke street, Dublin. *Trans.* 2.

1846 M'Ewen, William, M.D., Surgeon to Chester Castle; 27, Nicholas street, Chester.

1866 Macgowan, Alexander Thorburn, Kingswood park, near Bristol.

1823 †Macilwain, George, Consulting Surgeon to the Finsbury Dispensary, and to the St. Anne's Society's Schools; Matching, Harlow, Essex. C. 1829-30. V.P. 1848. *Trans.* 1.

1822 Macintosh, Richard, M.D.

1859 *M'Intyre, John, M.D., Odiham, Hants.

1873 MacKellar, Alexander Oberlin, M.S.I., Resident Assistant-Surgeon, St. Thomas's Hospital; Albert Embankment, Westminster Bridge.

1854 *Mackinder, Draper, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1860 Maclean, John, M.D., 24, Portman street, Portman square.

1849 Maclure, Duncan Maclachlan, M.B., Lecturer on Physiology at the Westminster Hospital; Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 34, Harley Street, Cavendish square.

1842 Macnaught, John, M.D., 74, Huskisson street, Liverpool.

1855 Marcet, William, M.D., F.R.S.; Villa Bianca, Cannes. C. 1871. *Trans.* 3. *Sci. Com.*

1867 Marsh, F. Howard, Assistant-Surgeon to St. Bartholomew's Hospital; 36, Bruton street, Berkeley square. *Trans.* 1.

1838 Marsh, Thomas Parr, M.D.

Elected

1851 MARSHALL, JOHN, F.R.S., *Vice-President*, Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; Examiner in Surgery at the University of London; 10, Savile row, Burlington gardens, C. 1866. V.P. 1875. *Trans.* 2.

1864 MASON, FRANCIS, Assistant-Surgeon to, and Lecturer on Anatomy at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. *Trans.* 1.

1869 MAYO, CHARLES, M.B., Colonial Surgeon at the Fiji Islands. [New University Club, St. James's street.]

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square.

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 11, Montpellier row, Blackheath, Kent.

1867 MEREDYTH, COLOMIATI, M.D., 76, Margaret street, Cavendish square.

1874 MERRIMAN, JOHN J., 45, Kensington square.

1852 MERRYWEATHER, JAMES, Consulting Surgeon to the National Dental Hospital; 25, Brook street, Grosvenor square.

1847 MERYON, EDWARD, M.D., F.G.S., 14, Clarges street, Piccadilly. L. 1859-60. C. 1864-5. V.P. 1868-9. *Trans.* 2.

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; 19, Temple row, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1873 MILNER, EDWARD, Surgical Registrar, St. Bartholomew's Hospital; 32, New Cavendish street, Portland place.

1863 MONRO, HENRY, M.D., Physician to St. Luke's Hospital; 13, Cavendish square. C. 1868.

Elected

1844 †MONTEFIORE, NATHANIEL, 36, Hyde park gardens.

1836 MOORE, GEORGE, M.D.

1873 MOORE, NORMAN, M.B., Lecturer on Comparative Anatomy, St. Bartholomew's Hospital.

1861 MOREHEAD, CHARLES, M.D., Hon. Surgeon to H.M. the Queen; Deputy-Inspector General of Hospitals; 11, North manor place, Edinburgh.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.B., Physician to the Manchester Royal Infirmary, and Lecturer on Medicine at the Manchester Royal School of Medicine; 1, St. Peter's square, Manchester.

1874 MORRIS, HENRY, M.A. Lond., Senior Assistant-Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place.

1851 MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General of Hospitals; Medical Inspector to the Local Government Board; and Member of the Senate of the University of Calcutta; 12, Durham villas, Kensington.

1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Clinical Medicine at, Guy's Hospital; 6, Finsbury Circus. *Trans.* 1.

1856 MURCHISON, CHARLES, M.D., LL.D. Edinb., F.R.S., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital, Consulting Physician to the London Fever Hospital; Examiner in Medicine at the University of London; 79, Wimpole street, Cavendish square. C. 1870-71. *Trans.* 3.

1875 MURPHY, WILLIAM KIRKPATRICK, M.A., M.D., 9, London street, Norfolk square, Hyde park.

1873 MURRAY, IVOR, M.D., F.R.S. Ed., The Knowle, Brenchley, Kent.

1863 MYERS, ARTHUR B. R., Surgeon to the 1st Battalion Coldstream Guards; Hospital, Vincent square, Westminster.

Elected

1859 NAYLER, GEORGE, Surgeon to the Hospital for Diseases of the Skin, Blackfriars; 3, Savile row, Burlington gardens.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park.

1843 †NEWTON, EDWARD, 4, Upper Wimpole street. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants.

1847 *NOURSE, WILLIAM EDWARD CHARLES, Surgeon to the Brighton Children's Hospital; Surgeon to St. Mary's Hospital, Brighton; 11, Marlborough place, Brighton.

1849 NOVERRE, ARTHUR, 16, Park street, Grosvenor square. C. 1870-71.

1864 NUNN, THOMAS WILLIAM, Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D., Mickleover, Derbyshire. *Trans.* 2.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1843 †O'CONNOR, WILLIAM, M.D., Senior Physician to the Royal Free Hospital; 30, Upper Montagu street, Montagu square.

1858 OGLE, JOHN WILLIAM, M.D., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; Inspector of Anatomy for the Provinces; 30, Cavendish square. C. 1873. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; 98, Friar Gate, Derby.

1860 OGLE, WILLIAM, M.D., Medical Officer of Health for East Hertfordshire; 10, Gordon street, Gordon square. S. 1868-70. *Trans.* 4.

Elected

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament Street].

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.

1873 ORD, WILLIAM MILLER, M.B., Assistant-Physician to, and Lecturer on Physiology at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 2.

1874 PAGE, HERBERT WILLIAM, M.B., M.C., 28, New Cavendish street.

1847 *PAGE, WILLIAM BOUSFIELD, Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.

1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., *President*, Sergeant-Surgeon Extraordinary to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-49. V.P. 1861. T. 1867. P. 1875. *Trans.* 9. *Sci. Com.*

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1847 PARKER, NICHOLAS, M.D., Paris.

1873 PARKER, ROBERT WILLIAM, 15, Bedford place, Russell square.

1841 PARKIN, JOHN, M.D., Rome. [53, Margaret street.]

1851 PART, JAMES, M.D., 89, Camden road, Camden town.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to, and Lecturer on Physiology at, Guy's Hospital; 35, Grosvenor street.

1869 PAYNE, JOSEPH FRANK, M.B., Assistant-Physician to, and Lecturer on Materia Medica at, St. Thomas's Hospital; 6, Savile row, Burlington gardens.

1845 PEACOCK, THOMAS BEVILL, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; Consulting Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 20, Finsbury circus. S. 1855-6. V.P. 1867. C. 1869. *Trans.* 2.

Elected

1856 PEIRCE, RICHARD KING, 16, Norland place, Notting hill.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 18, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.

1870 PERRIN, J. BESWICK, Medical Tutor, Royal School of Medicine; 10, Faulkner street, Manchester.

1852 PHILLIPS, RICHARD, 27, Leinster square, Bayswater.

1846 PHILP, FRANCIS RICHARD, M.D. [Colby House, Kensington.]

1867 PICK, THOMAS PICKERING, Assistant-Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; 7, South Eaton place, Eaton square. *Sci. Com.*

1851 *PICKFORD, JAMES HOLLINS, M.D., M.R.I.A., 1, Cavendish place, Brighton.

1841 †PITMAN, HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1.

1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L. 1859-62. V.P. 1870-1. *Trans.* 4.

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square.

1871 POORE, GEORGE VIVIAN, M.D., Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 30, Wimpole street.

Elected

1843 POPE, CHARLES, M.D., The Rectory, East Harptree, Bristol.

1846 POTTER, JEPHSON, M.D., F.L.S., 6, Soho street, Liverpool.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD. DOUGLAS, M.D., Senior Assistant-Physician to, and Lecturer on Materia Medica at, Charing Cross Hospital; Physician to the Hospital for Consumption, Brompton; 15, Henrietta street, Cavendish square.

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. *Sci. Com.*

1857 PRIESTLEY, WILLIAM OVEREND, M.D., Physician-Accoucheur to H.R.H. the Princess Louis of Hesse; Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. *Sci. Com.*

1869 PULLAR, ALFRED, M.D., Surgeon to the Kensington Dispensary; 47, Kensington park gardens.

1874 PURVES, WILLIAM LAIDLAW, M.D., Aural Surgeon to Guy's Hospital; 7, Hanover street, Hanover square.

1850 QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. *Trans.* 1. *Sci. Com.*

1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to the Eye Infirmary attached to the Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Trans.* 1. *Pro.* 2.

1852 RADCLIFFE, CHARLES BLAND, M.D., Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8.

1871 RALFE, CHARLES HENRY, M.D., M.A., Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.

Elected

1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital; the Pavement, Nottingham.

1869 READ, THOMAS LAURENCE, 57, Gloucester road, Queen's gate, South Kensington.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Trans.* 1.

1869 REEVES, WILLIAM, 5, the Crescent, Carlisle.

1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; 38, Grosvenor street. C. 1870.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

1847 RICHARDS, SAMUEL, M.D., 36, Bedford square.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., Warcop, Penrith.

1869 RICKARDS, WALTER, M.D., Physician to the Royal Free Hospital; 8, Cavendish place, Cavendish square.

1845 RIDGE, BENJAMIN, M.D., 21, Bruton street, Berkeley square.

1863 RINGER, SYDNEY, M.D., Professor of Materia Medica in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square.

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square. *Trans.* 2,

Elected

1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.

1852 ROBERTS, JOHN, M.R.C.P., the Park, Westow hill, Upper Norwood.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.

1843 ROBINSON, GEORGE, M.D. *Trans.* 2.

1843 RODEN, WILLIAM M.D., Morningside, Kidderminster, Worcestershire.

1829 ROOTS, WILLIAM SUDLOW, F.L.S., Surgeon to the Royal Establishment at Hampton Court; Canbury House, Kingston, Surrey.

1850 ROPER, GEORGE.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.

1849 ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1834 RUMSEY, HENRY WYLDBORE, M.D., F.R.S., Priory House, Cheltenham.

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital, and Professor of Medicine at Queen's College, Birmingham; 91, New Hall street, Birmingham.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S.E., Professor of Physiology in the University of Edinburgh.

Elected

1856 SALTER, S. JAMES A., F.R.S., F.L.S., Dental Surgeon to, and Lecturer on Dental Surgery at, Guy's Hospital; 17, New Broad street, City. C. 1871. *Trans.* 2.

1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3.

1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., Jodrell Professor of Human Physiology and Histology at University College, London; 49, Queen Anne street, Cavendish square. C. 1869-70. *Trans.* 2. *Sci. Com.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1847 SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Lecturer on Mental Diseases at University College, London; Sandywell park, Cheltenham.

1869 SANSOM, ARTHUR ERNEST, M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 29, Duncan terrace, Islington. *Trans.* 1.

1845 SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; Examiner in Surgery at the University of London; 66, Brook street, Grosvenor square. C. 1871-2. *Trans.* 4. *Sci. Com.* 3.

1873 SCOTT, J. M. JOHNSTON, M.D., 13, Eglinton place, Crumlin road, Belfast.

1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street. *Trans.* 2.

Elected

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1837 †SHARPEY, WILLIAM, M.D., F.R.S., LL.D., Member of the Senate of the University of London; 50, Torrington square. C. 1848-9. V.P. 1862.

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Trans.* 4.

1848 *SHEARMAN, EDWARD JAMES, M.D., F.R.S. Edin., F.L.S., Consulting Physician to the Rotherham Hospital; Moorgate, Rotherham, Yorkshire.

1859 SIBLEY, SEPTIMUS WILLIAM, 12, New Burlington street. *Trans.* 4. *Sci. Com.*

1849 SIBSON, FRANCIS, M.D., F.R.S., *Librarian*, Consulting Physician to St. Mary's Hospital; Member of the Senate of the University of London; 59, Brook street, Grosvenor square. C. 1863-4. L. 1873-5. *Trans.* 1. *Sci. Com.*

1848 SIEVEKING, EDWARD HENRY, M.D., Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. *Trans.* 2. *Sci. Com.*

1871 SILVER, ALEXANDER, M.D., Physician to, and Lecturer on Clinical Medicine at, Charing Cross Hospital; 2, Stafford street, Bond street.

1842 †SIMON, JOHN, D.C.L., F.R.S., Surgeon to St. Thomas's Hospital; Medical Officer of the Privy Council and of the Local Government Board; Whitehall, and 40, Kensington square. C. 1854-5. V.P. 1865. *Trans.* 1.

1865 SIMS, J. MARION, M.D., Surgeon to the New York State Women's Hospital; 267, Madison Avenue, New York.

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Nice.

Elected

1872 SMITH, GILBART, M.A., M.B., Physician to the Royal Hospital for Diseases of the Chest, City road; Visiting Physician to the Margaret Street Infirmary for Consumption; 68, Harley street, Cavendish square.

1866 SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 2, Portugal street, Grosvenor square.

1835 SMITH, JOHN GREGORY, 23, Gloucester place, Greenwich.

1838 †SMITH, SPENCER, Surgeon to, and Lecturer on Clinical Surgery at, St. Mary's Hospital; 9, Queen Anne street, Cavendish square. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. C. 1875. *Trans.* 3. *Sci. Com.*

1864 *SMITH, THOMAS HECKSTALL, Rowlands, St. Mary Cray, Kent.

1845 SMITH, WILLIAM, 70, Pembroke road, Clifton, Bristol. *Trans.* 1.

1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, Royal County Hospital, Winchester.

1868 SOLLY, SAMUEL EDWIN, Ivy House, East Sheen.

1865 SOUTHAM, GEORGE, Surgeon to the Manchester Royal Infirmary, and Lecturer on Surgery at the Manchester Royal School of Medicine; 10, Lever street, and Oakfield, Pendleton, Manchester. *Trans.* 4.

1865 SOUTHEY, REGINALD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 6, Harley street, Cavendish square.

Elected

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.

1874 SPARKS, EDWARD ISAAC, M.B., Lecturer on Skin Diseases at the Charing Cross Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; 46, Queen Anne street, Cavendish square. *Trans.* 1.

1851 SPITTA, ROBERT JOHN, M.B., Medical Officer to the Clapham General Dispensary; Clapham Common, Surrey. *Trans.* 1.

1875 SPITTA, EDMUND J., late Demonstrator of Anatomy at St. George's Hospital; Clapham Common.

1843 *SPRANGER, STEPHEN, Cape Town, South Africa.

1867 SQUAREY, CHARLES EDWARD, M.B. *Trans.* 2.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board. [Greenford House, Sutton, Surrey.]

1842 †STEWART, ALEXANDER PATRICK, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham.

1865 STOKES, WILLIAM, Jun., M.D., Professor of Surgery, Royal College of Surgeons, Ireland; Lecturer on Surgery at the Carmichael School of Medicine, and Surgeon to the Richmond Surgical Hospital; 3, Clare street, Merrion square, Dublin, *Trans.* 1.

1843 STORKS, ROBERT REEVE, Paris.

1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5.

1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.

1863 STURGES, OCTAVIUS, M.D., Assistant-Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square.

Elected

1871 SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1869 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.

1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health; Town Hall, Oldham.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

1854 *SYMONDS, FREDERICK, Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the Oxford Dispensary; 35, Beaumont street, Oxford.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 1.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to Guy's Hospital; 15, St. Thomas's street, Southwark.

1852 TAYLOR, ROBERT, Surgeon to the Central London Ophthalmic Hospital, and to the Cripples' Home, Marylebone road; 7, Lower Seymour street, Portman square.

1845 TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 1.

1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 3, Upper George street, Bryanston square. S. 1871-4. *Trans.* 1. *Sci. Com.*

1857 THOMPSON, HENRY, M.D., Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

Elected

1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 4.

1862 THOMPSON, REGINALD EDWARD, M.D., Assistant-Physician to the Hospital for Consumption, Brompton; 8, Cranley place, Onslow square. *Trans.* 1. *Sci. Com.*

1848 TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 60, Grosvenor street.

1872 TOMES, CHARLES S., B.A., Assistant-Surgeon to the Dental Hospital; 37, Cavendish square.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

1871 *TREND, THEOPHILUS W., M.R.C.P. Edinb., Raeberry Lodge, Southampton.

1867 TROTTER, JOHN WILLIAM, Assistant-Surgeon, Coldstream Guards; Hospital, Vincent square, Westminster.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, President of the Royal College of Surgeons of Ireland; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1855 TULLOCH, JAMES STEWART, M.D., 1, Pembridge place, Bayswater.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Resident-Assistant Physician, St. Thomas's Hospital.

1873 TURNER, GEORGE BROWN, M.D., Surgeon to the East Sussex Infirmary; 3, Warrior square, St. Leonard's-on-Sea.

1870 VENNING, EDGCOMBE, Assistant-Surgeon, 1st Life Guards; Knightsbridge Barracks, and 87, Sloane street.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 44A, Wimpole street, Cavendish square.

Elected

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to, and Lecturer on Medical Jurisprudence at, St. George's Hospital; 14, Park lane.

1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.

1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 25, Lowther street, Carlisle.

1867 *WALLIS, GEORGE, Benet street, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Demonstrator of Anatomy at St. Bartholomew's Hospital; 39, Weymouth street, Portland place.

1852 WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 37, Queen Anne street, Cavendish square. C. 1872. *Trans.* 1.

1851 WALTON, HAYNES, Surgeon to St. Mary's Hospital, and to the Ophthalmic Department; 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1818 WARE, JOHN, Clifton Down, near Bristol.

1866 WARING, EDWARD JOHN, M.D. [Agents: Messrs. Grindlay and Co., 55, Parliament street]; 49, Clifton gardens, Maida vale.

Elected

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Liverpool Northern Hospital, and Lecturer on Anatomy and Physiology in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.

1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic and to the Central London Ophthalmic Hospitals; 7, Henrietta street, Cavendish square. *Trans.* 1.

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Trans.* 6.

1835 †WEBSTER, JOHN, M.D., F.R.S., Physician to the Scottish Hospital, and Consulting Physician to the St. George's and St. James's Dispensary; 9, Queen street, St. Andrew's. C. 1843-4. V.P. 1855-6. *Trans.* 6. *Pro.* 1.

1844 †WEGG, WILLIAM, M.D., *Treasurer*, 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-5.

1874 WELLS, HARRY, M.D., British Vice-Consulate, Gualeguay-chu, Entre Rios, Argentine Confederation.

1861 WELLS, JOHN SOELBERG, Professor of Ophthalmology in King's College, London, and Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal London Ophthalmic Hospital; 16, Savile row.

1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. *Trans.* 9. *Pro.* 1.

Elected

1842 †WEST, CHARLES, M.D., 61, Wimpole street, Cavendish square. C. 1855-6. V.P. 1863. *Trans.* 2. *Sci. Com.*

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Assistant-Physician to St. George's Hospital; 37, Green street, Grosvenor square.

1849 WHITE, JOHN.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1844 †WILDBORE, FREDERIC, 245, Hackney road.

1870 *WILKIN, JOHN F., M.D. and M.C., The Leas, Folkestone.

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 77, Grosvenor street, Grosvenor square.

1863 WILLETT, ALFRED, Assistant-Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]. C. 1849-50. V.P. 1860-1. P. 1873-4. *Trans.* 1. *Sci. Com.*

1859 *WILLIAMS, CHARLES, Assistant-Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.D., Physician to the Hospital for Consumption, Brompton; 47, Upper Brook street, Grosvenor square. *Trans.* 3.

1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; Consulting Physician to the Royal Infirmary for Children and Women; 28, Harley street, Cavendish square.

Elected

1859 WILLIAMS, JOSEPH, M.D. [3, Chichester street, Upper Westbourne terrace.]

1868 WILLIAMS, WILLIAM RHYS, M.D., Lecturer on Mental Diseases at St. Thomas's Hospital; Bethlehem Royal Hospital, Lambeth road.

1829 WILLIS, ROBERT, M.D., Barnes, Surrey. L. 1838-41.

1839 †WILSON, ERASMUS, F.R.S., Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. *Trans.* 2.

1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 WISE, THOMAS ALEXANDER, M.D., Thornton, Beulah hill, Upper Norwood, Surrey.

1841 WOOD, GEORGE LEIGHTON, 27, Queen square, Bath.

1851 WOOD, JOHN, F.R.S., Surgeon to King's College Hospital, and Professor of Surgery in King's College, London; Examiner in Anatomy and Physiology at the University of Cambridge; 68, Wimpole street. C. 1867-8. *Trans.* 3.

1872 WOOD, SAMUEL, St. Mary's Court, Shrewsbury.

1848 WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8.

1842 WORTHINGTON, WILLIAM COLLINS, Senior Surgeon to the Lowestoft Infirmary; Lowestoft, Suffolk. *Trans.* 3.

1865 WOTTON, HENRY, 62, Bedford gardens, Kensington.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1853 BRODIE, SIR BENJAMIN COLLINS, Bart., M.A., D.C.L., F.R.S., Brockham Warren, Reigate.

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1873 CHRISTISON, SIR ROBERT, Bart., M.D., D.C.L., LL.D., Professor of Materia Medica in the University of Edinburgh; Physician-in-Ordinary to H M. the Queen in Scotland; 40, Moray place, Edinburgh.

1868 DARWIN, CHARLES, M.A., F.R.S., Corresponding Member of the Academies of Sciences of Berlin, Stockholm, Dresden, &c.; Down, Bromley, Kent.

1857 FARR, WILLIAM, M.D., D.C.L., F.R.S., General Register Office, Somerset House, and Southlands, Bickley, Kent.

1868 HOOKER, JOSEPH DALTON, M.D., D.C.L., LL.D., F.R.S., Director of the Royal Botanic Gardens, Kew; President of the Royal Society; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 26, Abbey place, St. John's wood.

Elected

1847 OWEN, RICHARD, D.C.L., LL.D., C.B., F.R.S., Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge; Secretary to the Royal Society, &c.; Lensfield Cottage, Cambridge.

1875 STOKES, WILLIAM, M.D., D.C.L., LL.D., F.R.S., Regius Professor of Physic at Dublin University; 5, Merrion square north, Dublin.

1868 TYNDALL, JOHN, LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1841 ANDRAL, G., M.D., Member of the Institute of France and of the Academy of Medicine; Paris.

1872 BERNARD, CLAUDE, Member of the Institute of France, and of the Academy of Medicine; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Rue de Luxembourg, 24, Paris.

1864 DONDERS, FRANZ CORNELIUS, M.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1875 DRAPER, JOHN WILLIAM, M.A., LL.D., Emeritus Professor of Chemistry and Physiology in the University of New York; 13, University Buildings, Washington square, New York.

1835 EKSTRÖMER, CARL JOHAN, M.D., C.M., K.P.S., and W., Physician to the King of Sweden; President of the College of Health, and Director-General of Hospitals; Stockholm.

1841 EHRENBERG, CHRISTIAN GOTTFRIED, Foreign Associate of the Academy of Sciences of the Institute of France, Berlin.

1868 GROSS, SAMUEL D., M.D., F.C.P. Philad., D.C.L. Oxon., Professor of Surgery in the Jefferson Medical College of Philadelphia.

1866 HANNOVER, ADOLPH, M.D., Professor at Copenhagen.

1873 HELMHOLTZ, H., Professor of Physics and Physiological Optics; Berlin.

1859 HENLE, J., M.D., Professor of Anatomy at Göttingen.

Elected

1873 HOFMANN, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

1868 KÖLLIKER, ALBERT, Professor of Anatomy at Würzburg.

1856 LANGENBECK, BERNHARD, M.D., Professor of Surgery in the University of Berlin.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1862 PIROGOFF, NIKOLAUS, M.D., Professor of Surgery to the Medico-Chirurgical Academy in St. Petersburg, and Director of the Anatomical Institute; Consulting Physician to the Hospitals Obuchow, Peter-Paul, and Maria Magdalena; St. Petersburg.

1875 PORTA, LUIGI, Professor of Medicine in the Royal University of Pavia; Member of the Royal Institute of Science and Literature of Lombardy, Milan, &c.; Pavia.

1850 ROKITANSKY, CARL, M.D., Curator of the Imperial Pathological Museum, and late Professor of the University of Vienna. Referee for Medical and University Education to the Austrian Ministry; Vienna.

1856 STROMEYER, LOUIS, M.D., Director-General of the Medical Department of the Army of Hanover; Hanover.

1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

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ADVERTISEMENT.

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CASE

OF A

FOREIGN BODY IMPACTED FOR TWENTY MONTHS IN THE FEMALE PELVIS.

RECOVERY AFTER ITS REMOVAL.

BY

RICHARD BARWELL, F.R.C.S.;

SURGEON TO CHARING CROSS HOSPITAL.

(Received September 13th—Read October 27th, 1874.)

At the latter end of May, 1874, I was called in consultation by Dr. ——, who told me the following history. In the middle of February, 1873, he had been consulted by Miss X—, on account of severe pains in the left hip, thigh, and leg, which were ascribed to sciatica. He, however, gave it as his opinion that the pains proceeded from some deep-seated cause of irritation, the nature of which he could not discover. He prescribed opium with frequent hot baths, which treatment so far relieved or cured the pains that his one visit sufficed and for nearly a year he lost sight of his patient.

In January of the present year he was again called in and found the patient suffering from an abscess over the left hip. He reverted to his previous theory of deep-seated local irritation. Miss X— asked if anything introduced "into her body" could cause such symptoms, and Dr. ——, seizing this clue as basis for cross-questioning, extracted, with great difficulty, the following history:

Miss X— had procured the attendance of an abortionist calling himself Dr. Cooke. This man came to her at the latter end of 1872, and passed "into her body" an instrument, which, from her description, Dr. —— judged to be an elastic catheter. This instrument he left *in sitû*, promising to call the following day. Some hours after this procedure Miss X— had occasion to go to the water-closet and had a hard, somewhat difficult motion, during the passage of which she imagined that the instrument was slipping downwards, and she pushed it with her hand further into the vagina.

The next morning the man came again and found no part of his instrument except "the little ivory button at the end." He is stated to have been much alarmed, but, learning that his victim had been to the water-closet, told her that she must have passed it there. But Miss X— says that before leaving the closet she put on a diaper, that the instrument was there when she did so, and that she never found it in her bed nor in any of her clothing.

I found the patient, twenty-four years old, somewhat emaciated and pale, but not exhausted, although feeble. The left limb was thinner than the other, with apparent lengthening, as in the first stage of hip disease, but there was nothing amiss with that joint. Over the right trochanter was a large abscess, the covering of which was red over a space nearly as large as the hand, and here the skin was thin. In front of the tuber ischii was the mouth of a sinus with everted edges and pouting granulations; a long probe, passed into this, went up a considerable distance by the side of the bowel. This sinus did not communicate with the abscess.

Chloroform was administered. Before opening the abscess I examined both rectum and vagina, but only made out that both cavities were surrounded by pus.

Dr. —— had an idea that the foreign body, whatever it might be, had passed into the abscess, and there certainly was a hard line or spot in its midst. I therefore made an incision where the skin was thinnest and where this spot could be easily reached, and let out a full pint of most horribly offensive, feculent pus, with which came also a

quantity of gas, doubtless intestinal. I passed my finger into the wound, but found no foreign body; the hard spot was simply a portion of undissolved tissue forming a sort of buttress or bridge between the outer and inner walls of the abscess. The place of my incision had been chosen with reference to this spot, not to the outflow of pus. I therefore made another opening at the most dependent part possible.

On 28th May we met again with the object of further examination. Miss X— was greatly improved both in strength and spirits; indeed, was so hopeful that she said to Dr. —— she believed the foreign body must have disappeared, or that it may after all have passed from her when she went to the closet as before related; the difference in her feelings of health appeared to prompt this change of view. The abscess was contracting, but it still discharged a pus, that now was only slightly odorous.

I first examined the vagina; the speculum showed nothing save plentiful leucorrhœa, both vaginal and uterine, a long, pendent *cervix*, a patent and ragged *os uteri* somewhat congested. Digital examination revealed the same state of parts; no wound, no cicatrix, could be detected. I passed the finger quite round the cervix, and in doing so found at the back of the posterior *cul-de-sac*, quite at its upper angle, a rather hard band which ran transversely across this part of the cavity, behind its mucous membrane. I could trace from one side to the other the curve of this band, which was in shape and direction exactly like the ilio-pectineal line, distinguishable, however, by its position too near the vagina, by its yielding slightly on pressure, but, perhaps, chiefly by the fact that it was not within reach of the finger except when strong pressure was made over the pubes, at which time it could be felt to descend within touch. Nevertheless, there was no evidence that this line of hardening was due to the presence of a foreign body; it might be, and indeed at this time I inclined to the belief that it was, the thick limiting membrane at the top of the abscess, which, when reflected from the back to the front, would present

itself to the finger as a fold, and would appear, therefore, like a distinct edge.

I now explored the rectum and could for some time find nothing abnormal, save that the cavity seemed to take a direction hardly enough to the left. Guided by this, and thinking that the bowel was probably pushed over by the abscess, I directed much attention to the left side, but found nothing abnormal. I now sought diligently for the thickened band felt from the vagina, which, if my view of its position were correct, ought to lie in front of the rectum. After a time, by making considerable pressure over the pubes I brought within touch a transverse band of induration, which appeared *convex* towards the gut. I now, therefore, for the first time felt a distinct belief in the presence of a foreign body, and I examined the mucous membrane with great care, since the contents of the abscess proved that there must be an opening in the intestine somewhere. I found none either upon or below this induration; but I detected a part, a little on the right, where was a greater amount of thickening running upwards out of reach; I was determined to trace this, if possible, to its end, and made more and more pressure with my right hand over the pubes, and at last so much that I had to make counter-pressure with my knee against the sacrum. In that posture I traced this ascending induration to its end, and just at that end I found an opening like a pouch or diverticulum, into which I passed the tip of my forefinger, and crooking it a little downwards came across a rough substance which felt like a portion of a narrow cylinder running transverse to the axis of the pelvis.

I told Dr. —— that I had found the foreign body, and in a few words of rapid consultation it was agreed that I should endeavour to extract it without chloroform, if the patient could bear it, since I was very unwilling to risk, in the struggles and kicking which chloroform induces, the loss of a foreign body I had taken so much pains to find.

I therefore, keeping my finger tip on the substance, introduced a long, rather narrow pair of polypus forceps and

succeeded in insinuating one of the blades beneath the body, grasped it with but little pressure and began to draw it cautiously down; soon, however, a sense of crackling communicated through the instrument warned me of possible breakage, but by this time I had drawn the foreign body somewhat down and into the form of a loop. Into this loop I crooked my finger tip and drew cautiously and slowly into the bowel, and so out through the anus, the instrument now on the table, viz. a gum-elastic catheter, apparently a No. 7 or 8, which had lain for twenty months in the cellular tissue between the uterus and rectum.[1] I need only add that the body was not hard and stiff as now, for it could not in such condition have been safely removed in the manner described; it was, on the contrary, completely sodden and as flexible as a strip of wash leather, so that it folded up into a loop with the greatest ease, the middle portion coming down first and being followed by both ends.

On the fourth day after this operation I heard from Dr. —— that the patient had not had a bad symptom.

It appears to me that this case is to be read in the following way. The abortion procurer probably introduced the catheter, with the stilette in it, through the os uteri; but being ignorant of the direction of this organ, sloping from below forward as well as upward, he thrust the instrument straight onward through the back of the womb and into the cellular tissue behind it. The occurrence of bleeding would lead him to suppose his object effected, and he removed the stilette, most likely, after the manner of blunderers, not by simple withdrawal of the wire, but also by pushing the instrument off it further into the wound. Subsequent soaking in warm discharges and blood fully accounts for the loosening of the bone extremity.

Some obscurity hangs of necessity about the next phase of the history; it is impossible to say how far the patient may have pushed the instrument through the opening,

[1] This catheter is now in the Museum of the Royal College of Surgeons, Pathological Series, 1131B.

neither is it known whether on the next morning the abortionist searched in the vagina for the catheter. Therefore it is not on record how soon the whole body of the instrument became embedded in the tissues, nor can I with certainty specify the mechanism whereby such inception took place. It is, however, barely possible that the unhappy girl could herself have intruded the whole length of the catheter, since this would connote the introduction of her finger up to the os uteri; nevertheless, she probably pushed it a considerable distance, and the inglutition was completed by contractions of the uterus, doubtless pretty active, aided, perhaps by movements of the rectum in defecation.

However that may be, the position of the instrument was, when I found it, very singular; it seemed to be in front of the brim of the pelvis, curled up so as to imitate its curve with great precision; the middle was between the body of the uterus and the rectum, the ends probably close to the bone on either side.

The rest of the history is far plainer. The presence of this body produced violent irritation and some inflammation (perimetritis), during which the severe pains, ascribed by Dr. —— with so much acumen to deep local irritation, manifested themselves. The inflammation went on to suppuration; matter gathered around the rectum, some finding its way downward by the side of that cavity, and producing the fistulous opening in front of the tuber ischii; but most of it, since she was much confined to bed, passing to the back of the intestine, and, following the course of the pyriformis muscle, made exit through the sciatic foramen to the hip, not communicating with the track of the sinus.

There is very little evidence to show at what period the abscess burst into the rectum, but probably not very long before I saw her, as her health would, I think, have suffered more materially had so foul an abscess as that which I opened been of very old standing.

The patient continued to pass a little flatus through the abscess and wound on the hip for ten days; this then ceased. In the beginning of July the wounds and sinus had healed. She remains perfectly well at the present time.

CASES

OF

SYPHILITIC REINFECTION.

WITH REMARKS.

BY

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(Received October 20th—Read November 24th, 1874.)

It was long believed that a person who had once been the subject of syphilis was thereby rendered more prone to contract again this disease, and that if subsequently exposed to contagion he would be liable to acquire one attack of syphilis after another. It was further considered that with each new infection fresh poison was absorbed into the system, adding to the virulence of that already lurking there, so as to stimulate and intensify the existing disease.

Such were the current opinions until Ricord taught that syphilis, like smallpox and other disorders of this class, exerts such an effect upon the economy as to protect it from further contamination. This condition he described as a *diathesis* which, as long as it lasts, confers complete immunity from another infection, and renders the person incapable of developing either an indurated chancre or other constitutional symptom. In his experience this protective influence terminates only with the life of the patient, but he admits that by ana-

logy the syphilitic diathesis should in certain subjects wear out and become extinguished, so as to permit a repetition of the disease, just as recurrences of measles, scarlet fever, &c., are occasionally, though exceptionally, met with. At that date, however, he had never known an instance of syphilitic reinfection, and stated that "science does not yet possess a single well-proved example of a second attack of syphilis."[1]

In consequence of this statement Ricord has been credited with the dogma that a second infection of syphilis is impossible; but, far from this being the case, he distinctly says—"It is not that I deny the possibility of a repetition of the indurated chancre; on the contrary, *I believe in it*, and I believe in it firmly, although clinical experience has as yet refused me proofs of it."[2]

The sagacity of Ricord's perceptions on this subject have now received ample confirmation, not only as regards the persistence of the syphilitic diathesis, which generally lasts for many years, and often, indeed, until the end of life; but also as to the occasional extinction of this peculiar influence, so that the patient may regain his former aptitude to contract the disease, for it has been conclusively demonstrated that syphilis may be repeated in the same person,[3]

The instances recorded amount to a considerable number, but they are mostly scattered throughout the medical periodicals in such a manner as to render them unavailable for general use, and hence they are scarcely known to the profession. I have therefore appended a list of the cases that I have been able to find, with their references, and now proceed to give the details of eleven others which have passed under my own observation.

[1] 'Leçons sur le Chancre,' 1858, p. 159.

[2] Loc. cit., p. 158.

[3] Ricord, in a letter to Mr. Acton, published in the 'British Medica Journal,' 1872, vol. ii, p. 228, now fully admits this, as shown by the following sentence:—"Now, as we have authentic examples of fresh contagions of indurated chancre, with consecutive evolutions of the whole series of constitutional symptoms, this proves that the patients have been cured; just as the possibility of contracting smallpox afresh, or of vaccination again taking, proves that the first variolous or vaccine influence has ceased."

With the exception of one of these cases, the rest have occurred in my private practice; and although I could give the particulars of several which have come before me at the hospital and elsewhere, sufficiently convincing to myself as examples of syphilitic reinfection, I have purposely excluded them as failing in some respects to satisfy the just requirements of others, and have selected those only which appear to me to be beyond dispute. Of these eleven cases I have myself treated seven (Nos. 1, 2, 3, 4, 5, 8, 10) for both infections. In three (Nos. 6, 9, 11) the former syphilis is guaranteed by competent medical witnesses; whilst the only one (No. 7) in which there is no evidence of a second infection beyond the patient's statement occurred in a man of great intelligence, who had had some amount of medical reading, and, from my personal knowledge of him, I cannot doubt his veracity or the accuracy of his story.

Case 1.—November 7th, 1863. A gentleman, æt. 28, in good health, consulted me for a large typically indurated chancre on the corona and glans, with indurated inguinal glands. He had a prejudice against mercury and would not take it for five or six weeks, when, the chancre having increased to nearly the size of a florin and showing no disposition to heal, he commenced a mercurial treatment. In a week the improvement was so marked that all reluctance ceased, and he continued to take the medicine for three or four months, until the induration had quite disappeared. Except a slight sore throat of a doubtful character, there were no constitutional symptoms.

On November 16th, 1867, he showed me an abrasion with a very suspicious base on the inside of the prepuce, the result of connection a week ago, which developed into an indurated chancre; two other sores also formed and indurated. The inguinal glands could be felt separate and slightly enlarged, but they could not be distinctly made out owing to the large amount of fat in the groins. The sores soon healed under mercury, but the induration did not pass away until the end of February. On May 25th a large mass

of induration had formed at the frænum, and gradually extended half-way around the corona and glans. Much deposit of similar nature had also taken place in the lymphatics on the dorsum of the penis, quite to its root; these hardenings gradually yielded to mercury. Subsequently (October 9th) a gumma formed on the inside of the right leg, which broke, leaving a very obstinate sore; another appeared, but subsided. A few months after this a tertiary ulcer occurred on the leg, and six months later a deposit again took place in the lymphatics of the penis; this symptom was very persistent, and recurred two or three times. His health throughout was good. He has since remained free from disease, and is now the father of two healthy children.

CASE 2.—February 16th, 1864. A farmer, æt. 36, of robust health, covered with a copious eruption of roseola and lichen, following a sore at the corona which is now healed, though a slight induration can still be felt. Under mercurial treatment all symptoms disappeared, and he remained free from disease until—

November 12th, 1870, when he came to me with a severe eruption of roseola and psoriasis, and mucous papules of the throat. He said that after intercourse in July a sore formed on the inside of the foreskin, which healed without treatment about the end of August. The eruption began to appear three weeks ago. Under mercury these symptoms passed away, but he had several relapses in the form of psoriasis, mucous papules of the throat and anus, superficial ulcerations on the trunk, scrotum, &c., which required treatment at intervals until the summer of 1873. He has since continued well.

CASE 3.—June, 1864. A healthy man, æt. 22, contracted a chancre three months ago, which has healed; has now severely ulcerated mucous papules of the throat, fauces, and palate, very extensive and very persistent, with a fading eruption of roseola and psoriasis. He recovered under mercurial treatment, and remained free from all trace of disease until—

May 9th, 1873, when he showed me three indurated chancres on the inside of the foreskin, the result of connection about a fortnight since. Each sore was situated on a well-defined induration, and could be picked up between the fingers as a small button-like mass. The inguinal glands were not affected. Mercury was given. On the 24th the sores were nearly well, but the induration was still quite perceptible. I have not heard of him since, though he promised to inform me if anything else occurred.

Case 4.—January, 1865. A healthy man, æt. 34. General roseola, with universal enlargement of glands, the result of infection probably in August 1864. There had been great pain and aching in the limbs previous to the appearance of eruption. He had had no treatment. In April he had very severe psoriasis of the trunk and limbs, followed by double iritis and great impairment of health. He now commenced mercury, but took it very irregularly. He continued in a cachectic state, having at intervals double orchitis, ulcers of the leg, and nodes, until 1870, since when there have been no visible manifestations of the disease; his health, however, has remained much enfeebled, and he has suffered from a lung affection considered to be of syphilitic origin.

In February, 1873, a large indurated chancre formed on the left thumb; it commenced as a pustule, and was contracted on January 24th from a patient who had two suppurating indurated sores on the foreskin, which were followed by roseola. The induration on the thumb was well marked, and the lymphatics over the metacarpal bone became swollen, hard, and tender on pressure, but neither the epitrochlear nor the axillary glands were affected. During March there was much nocturnal aching of the legs, but no other symptoms. No treatment.

Case 5.—August 12th, 1865. Clerk, æt. 32, thin, delicate-looking man. A very large indurated chancre at the corona, involving the glans and prepuce, inflamed and sloughing from

the application of nitrate of silver; inguinal glands much enlarged and painful. Mercury was given when the inflammation had subsided. He had subsequently a roseola and sore throat, on recovery from which he remained free from disease until—

August 3rd, 1871, when he came to me with a small indurated chancre on the dorsum of the penis, which commenced as "a pimple" after connection a week ago.

On the 7th the sore was as large as a threepenny piece and the induration well marked; the glands in the groins were enlarged, one on the left side ultimately suppurated. I gave him mercury. At the beginning of September the sore and bubo were nearly healed, and he ceased to attend.

Case 6.—May 25th, 1866. A fine healthy man, æt. 30. Indurated chancre on inside of prepuce, which commenced as a pustule directly after intercourse on April 7th, and another on the opposite side of the foreskin which appeared a fortnight after the first; there were also cicatrices of two or three other sores; inguinal glands indurated, one on each side soft and painful; patches of roseola on shoulders and arms. Has been taking iodide of potassium for more than a month. Under mercurial treatment the sores healed and the eruption disappeared.

The patient told me that in 1857 he had a chancre followed by secondary eruption, and in 1858 severe palmar psoriasis; was treated actively with mercury on each occasion, and has remained well until now. This statement was confirmed by the medical man who had previously attended him.

Case 7.—October 17th, 1866. Journalist, æt. 24; strumous habit; family phthisical. Scattered pustular eruption of face and trunk; mucous papules on lips, cheeks, and throat; induration perceptible at corona; inguinal glands indurated. Contracted a chancre five weeks ago when travelling, and has had no treatment. He recovered slowly under mercury, and I did not see him again until March 21st,

1871, when he presented himself with a general pustular eruption of the face, trunk, and limbs, and in a very cachectic condition. He had been shut up in Paris during the siege, and had lived very badly. He told me that he had kept free from disease until the end of 1868, when he got another chancre, which was followed by a severe eruption; under medical advice in Germany and Paris he took mercury for several months, and had no relapse until these spots began to appear about six weeks ago. I again gave mercury with tonics. He soon improved, and has remained well up to the present time.

Case 8.—July 10th, 1868. House decorator, æt. 28; healthy man; indurated chancre at meatus, extending nearly half an inch down the urethra, so as to diminish sensibly the stream of urine; induration very marked; glands in groins indurated, one on each side tender on pressure. Noticed the sore ten days after connection, and has been under treatment for six weeks. He gradually got well with mercury, but required the use of a bougie for several months to keep the meatus dilated. He had no constitutional symptoms.

On July 30th, 1870, he again consulted me for a well-marked indurated chancre on the inside of the foreskin, with cicatrices of other sores, and urethral discharge. The glands in the groins were indurated; spots of psoriasis were scattered over the trunk and limbs, which first appeared about three weeks ago, and the fauces were covered with mucous papules. The infecting intercourse occurred about three months ago, and he has had no treatment. Under mercury he rapidly got better, but he had several relapses, necessitating treatment for nearly a year, his throat being especially troublesome.

December 7th, 1872.—He showed me a chancre at the upper part of the corona, the result of intercourse ten days ago; the base was distinctly indurated; the inguinal glands were not affected. The sore continued to spread until mercury was given, when it slowly healed. During treatment mucous papules came out on the cheeks and fauces;

the inguinal glands also enlarged, and one in the left groin suppurated. I saw him in the summer of 1873; he was then quite well, and had had no eruption.

CASE 9.—October 19th, 1868. A healthy man, æt. 26. Indurated chancre on inside of prepuce, nearly as large as a threepenny piece, on a base which can be raised like a button between the finger and thumb. The intercourse occurred ten days ago, and he noticed an abrasion four days afterwards, to which he applied nitric acid and latterly bluestone. The sore is inflamed and suppurating freely; the inguinal glands are not generally affected, but one on the left side is large and tender. He would not take mercury, as he wanted "to see if a man could have secondaries as well as an indurated chancre twice." The chancre soon healed, but induration was very perceptible on December 3rd. I have not seen him since. He told me that six years ago he had an indurated chancre at the corona followed by severe eruption, for which he was treated with mercury, and has had no return of the disease.

CASE 10.—April 13th, 1871. A healthy man, æt. 22. Indurated chancre at frænum, another at corona, both suppurating; inguinal glands indurated. Contracted disease eight or nine weeks ago; had a crop of sores around corona, which healed, but about a month ago these two reulcerated. Has been taking mercury in small doses throughout. I gave him larger doses, even to sixteen grains of blue pill daily, but without affecting his gums in the slightest degree. The chancres healed, but on May 20th they again opened, and extensive mucous papules appeared on his tonsils and fauces.

July 11th.—The induration had completely disappeared, and his throat was well. He had no further symptom of disease until—

April 18th, 1873, when he showed me an abrasion at the corona following intercourse three days ago.

21st.—The abrasion had spread, and its base was suspiciously hard.

23rd.—Chancre with well-marked induration was now developed; the inguinal glands were not affected.

Mercury soon produced its effects upon the gums, and on May 1st the sore had healed, but the induration could be felt for two or three weeks.

In October, 1874, he again came under my care for a small non-indurated chancre, with suppurating buboes, when he told me that in the summer and autumn of 1873 he was under treatment for several weeks with "a syphilitic sore throat."

Case 11.—January 27th, 1874. Female, æt. 20; readmitted into the Lock Hospital for an ulceration of the os uteri and utero-vaginal discharge. There was a distinct induration on the margin of the right labium, the sore having just healed; glands in both groins markedly indurated; mucous papules on the tonsils and fauces; tongue fissured and sore at edges. Under mercury the induration and other symptoms subsided. No reliable history could be obtained as to the present infection except that the sore had existed to her knowledge for two or three weeks.

On April 4th, 1872, this patient was admitted into the Lock Hospital under my colleague Mr. James Lane. The case-book records that she had a large ulcerating sore on each side of the vaginal orifice, with a deep foul ulcer at the anus and indurated inguinal glands, followed by psoriasis. She was treated with mercury.

From the above cases it will be seen that the interval which occurred between the two infections varied from a year and nine months (No. 11) to more than nine years (No. 6), and that the severity of the second attack bore no relation whatever to that of the first, nor yet to the length of time which had elapsed since the previous disease.

Of the ten cases in which the first contagion gave rise to general syphilis, in four (Nos. 3, 4, 5, 9) the reinfection

showed itself in the form of an indurated chancre only, but in the other six (Nos. 2, 6, 7, 8, 10, 11) undoubted constitutional symptoms again occurred, and in one case (No. 2) much more severely than at the first attack six and a half years previously.

In the case (No. 1) where the first disease was limited to an indurated chancre with inguinal adenopathy, the reinfection was followed by the tertiary form of syphilis, without the intervention of any secondary affection.

It may be urged that this case cannot be considered an example of syphilitic reinfection, as no general symptoms followed the first contagion; but if a sore with induration of its base and associate glands be regarded as syphilis, this objection cannot hold, for induration was present in such a marked degree that it would have served as a typical case of indurated chancre. If, therefore, we admit with M. Follin that "an indurated chancre with its adenopathy is, indeed, already constitutional syphilis"[1]—and few, I think, will dispute the position—this case must be admitted into the list of second contagions.

The induration which forms at the base of a chancre is, I consider, proof that the disease has ceased to be local and has become general. It is the first expression of a constitutional taint which manifests itself at the point of inoculation, and therefore is not one of the local processes of primary syphilitic ulceration. If this view be correct induration is the earliest of the secondary symptoms, and its presence is as pathognomonic of a general syphilitic contamination as eruption or mucous tubercles.

The above case also illustrates what I have observed in others—that where the induration has been very great the eruptive stage would generally be slight, or sometimes even altogether omitted. It is as if the syphilis had in such instances run its course or become exhausted in developing this one symptom of induration, just as we occasionally see that with a transient roseola or slight outbreak of mucous tubercles the disease will come to an end and give rise to no further

[1] 'Traité de Pathologie Externe,' t. i, p. 740, 1861.

manifestations. My experience does not accord with those writers who state that a large amount of induration is a certain prelude to a severe attack of constitutional disease, but rather the reverse.

Another peculiarity attended this patient at his second contagion—tertiary lesions followed upon the chancre without the intervention of any of the so-called secondary affections. I have seen this previously in a few cases, but, except in the present instance, it has always been in strumous persons or in those whose health was much enfeebled.

The eighth case is especially interesting as showing the extraordinary receptivity of the patient for the syphilitic poison. In July, 1868, he had a very large indurated chancre of the meatus and urethra, which lasted many weeks; inguinal adenopathy was well marked, but he had no general manifestations. In July, 1870, he contracted other indurated chancres with implication of the glands, followed by persistent eruption and mucous papules. In December, 1872, he again contracted an indurated chancre attended by glandular swelling in the groins and mucous papules, thus showing three separate infections within four years. It is the only instance of the kind I have met with; but Diday, in his very interesting monograph on this subject, mentions a patient in whom four distinct infections occurred,[1] and Mr. Hutchinson has related an example of three separate contagions in the same individual.[2]

Remarks.—These cases of syphilitic reinfection are of much practical value in showing that the effects of syphilis upon the system are not of necessity life-long; but that the diathesis, or *intoxication*, as some prefer to call it, created by the disease may gradually wear out and become extinct so as to allow another infection. For we cannot admit that a

[1] 'De la Réinfection Syphilitique', Obs. xiv. See also 'Gazette Hebdomadaire,' 1855, p. 238.

[2] "When and how to use Mercury in Syphilis," 'Lancet,' 1874, vol. i, p. 157.

person already under the influence of syphilis can suffer another general contamination, and thus become the subject of two concurrent attacks of the same disease, each of which must be passing through a different stage of development.

The possibility of reinfection proves also that a syphilitic person may entirely regain his former condition of health, and, therefore, become the father of healthy offspring. Thus is explained a circumstance with which we have long been familiar, that persons whom we know to have been the subjects of syphilis will apparently recover from it, remain free from recurrence for the rest of life, and beget healthy children without a trace of hereditary disease. Such instances, and happily they are not rare, would lead to the conjecture that the disease cannot be merely in abeyance, and biding its time for a fitting opportunity to again break forth, but that it has been completely got rid of; and the fact that a second infection is possible proves that such may really be the case. Nor would it seem that instances of recovery from syphilis are very uncommon, for we have clinical evidence of it in such observations as those just alluded to, whilst the published cases of a second contagion now amount to a considerable number.[1]

These cases of syphilitic reinfection bear also with peculiar significance upon a much debated, though most important, question—viz. *the curability of syphilis*. In the instances here related, as well as in most of those recorded by others, a full treatment by mercury had been adopted, thus affording, should it still be really needed, additional testimony to the therapeutic value of that drug in syphilis, and confirming the opinion of those who state, as the result of long experience, that a prolonged course of mercury is the most certain method of preventing the more severe, as

[1] The cases which form the subject of this communication are, I believe, the first which have been recorded in this country, but the accompanying table shows as many as sixty which have been collected from various publications.

well as the more remote, lesions of syphilis. Great encouragement is, therefore, given to persevere in this mode of treatment, inasmuch as it offers a reasonable hope of *curing* the disease.

Many other points of interest are opened by these cases, to some of which I will refer, though they have but an indirect bearing upon our present subject.

They seem to throw much light on the real import of the induration which usually accompanies an infecting chancre, and is regarded as a necessary part of primary syphilitic ulceration rather than as one of the manifestations of general syphilis. When inoculation of an indurated chancre succeeds upon a person already the subject of syphilis, an ulcer with a *soft* base is produced, which in no respect differs from an ordinary non-indurated chancre, either in its appearance or behaviour; but, although no induration attends this sore, its real nature is in no respect altered, for when transmitted to a healthy individual a chancre forms which becomes indurated and gives rise to constitutional disease. Here, then, we see that although an infected person cannot escape the local action of the syphilitic poison—as evidenced by the formation of an ulcer—yet no induration is produced by it; a circumstance which, to my mind, shows conclusively that this symptom is not one of the local processes essential to the development of a particular form of chancre, but that it entirely depends upon a constitutional cause, which is unable to manifest itself as usual at the point of inoculation, in consequence of the economy being already under the influence of a former contamination.

The presence, then, of induration about a sore shows in the first place that the system was free from a previous syphilitic taint; and, in the second, that the patient is under the influence of a general syphilis of recent date; so that the formation of an indurated chancre in a person who has formerly had syphilis proves that he must have completely recovered from the foregoing attack.

Again, that induration is a constitutional symptom, and

not a local phenomenon, is seen in the fact that a person who has an indurated sore, but is as yet without any other manifestation of syphilis, will, if exposed to contagion, contract a chancre with a soft base, but not another indurated sore; and the same result obtains from the spontaneous or artificial auto-inoculation of the original chancre.

I claim, then, on the above grounds, that the induration at the base of a chancre is as certain evidence of a general contamination as any of the recognised secondary symptoms, and that when a chancre has become indurated it has ceased to be a primary affection.

Another thing which may be learnt from the cases just related is that a period of incubation does not necessarily precede the evolution of an indurated sore, for in most of them the chancre which initiated the second disease followed directly upon intercourse. This shows, I think, that the contagion must have been acquired from a primary sore which was still suppurating and in full activity, rather than from an indurated chancre which had ceased to suppurate, or from that very common cause of infection—mucous tubercles; in other words, that the disease must have been received from a *primary*, and not from a *secondary*, lesion of syphilis.

As long as the local processes are active about a chancre and it continues to suppurate, it is highly virulent and capable of ready inoculation, producing at once a pustule, succeeded by an ulcer, which in the case of the infecting chancre usually begins to indurate about the fifth to the eighth day. The inguinal glands take on a similar action about the same time, and as the chancre becomes indurated it passes into an indolent condition and ceases to form pus.

When the serous secretion of such a sore is inoculated it no longer gives rise to the same phenomena as those which follow the insertion of chancrous pus, but they are so precisely analogous to those which have been observed to attend the inoculation of mucous tubercles or the blood of a syphilitic person, by direct experiment, that the same description will

suffice. In both, a little reddening follows the puncture, which soon dies away, but after a period varying from three to six weeks a hardened papule begins to form at the point of insertion; this slowly enlarges, desquamates on its surface, and finally ulcerates. The glands also become simultaneously affected.

Both these modes of development, which have been seen to attend artificial inoculation, are met with in practice as the result of physiological contact. In some cases the sore follows immediately after connection, whilst in others there is a delay of varying length, known as the *period of incubation*, before the formation of the ulcer. When incubation occurs it shows that the infection has been received from some constitutional lesion of syphilis, or from an indolent non-suppurating indurated sore, which, as before stated, I look upon as a truly secondary affection. When, however, the chancre follows at once upon intercourse, its origin may be assigned to an active suppurating sore of recent date—a *true primary chancre*.[1]

In the one instance there is afforded an illustration of *secondary*, and in the other of *primary* syphilitic inoculation by natural means; and the resulting chancres at first differ as much in their appearance and conduct as in their mode of evolution, although the sore which occurs from primary inoculation may gradually lose its characteristics, and, becoming indolent, assume those of the chancre which is produced by the inoculation of secondary syphilis.

The propagation of syphilis is, I believe, greatly due to contagion imparted during its secondary stage, and hence the frequency with which examples of secondary inoculation—those in which a period of incubation is present—are met; so much so, indeed, that some modern authors altogether deny the *début* of syphilis by a pustule. The older writers, however, speak of this as the usual commencement of syphilis, and certainly clinical observation will lend testimony to its occurrence in a number of cases. A primary chancre gives rise to pain,

[1] I have had opportunity of verifying this statement in three or four instances by an examination of both the persons concerned.

and attracts the attention of the patient, who perforce abstains from intercourse for a time, and seeks relief for it. The secondary contagious affections, such as mucous tubercles, on the other hand, are generally painless and often pass unnoticed, so that the person continues to spread disease in ignorance of his dangerous condition.

The syphilitic poison loses in virulence and in its ready inoculability with the lapse of time, until at length a period arrives when it ceases to be communicable. The secretion, therefore, of an active primary sore is more powerfully contagious than that of a secondary affection, whilst that of the more advanced lesions is altogether incapable of inoculation. It would seem also that persons who have had syphilis, instead of being more liable to contract again this disease, as was formerly believed, are in reality less so; their receptivity apparently having been diminished by the former attack, and a stronger virus may therefore be required to infect such individuals than in the case of those who have never suffered, and who are consequently susceptible to the influence of a weaker poison. A person who has not had syphilis may, therefore, become contaminated by a secondary accident which would prove powerless to infect one who has had the disease; and thus, perhaps, a partial explanation may be afforded why in these examples of syphilitic reinfection a period of incubation does not more often precede the development of the chancre, since it is not improbable that the contagion has been acquired from a primary syphilitic ulcer, in which case the sore would follow immediately upon intercourse.

The comparative rarity of cases of second infection may perhaps to some extent be referable to the stronger virus of a primary chancre being necessary to excite a general reaction when the feebler virus of mucous tubercles would fail to do so; or it may be that the secretion of these latter is too weak to produce more in such individuals than a slight manifestation at the point of insertion which is overlooked.

The reason why in so many instances the disease has been

limited to an indurated chancre may possibly be that, although the former diathesis was sufficiently extinguished to allow another infection to take place, yet the system had not regained its normal condition sufficiently to permit an outbreak of general disease, and consequently the effect of the poison was limited to ulceration at the point of insertion, about which induration, the first and slightest of the constitutional symptoms, was developed. But that these chancres, even when they do not give rise to any other symptom than induration, are powerfully infecting and capable of causing severe disease in a healthy subject, has been shown by Diday in a case which occurred in his own practice; and it has further been proved that such chancres have been derived from specifically indurated sores which were followed by constitutional disease in the persons transmitting them.[1]

Some of these cases of second contagion aid also in determining the position which certain of the manifestations known as *tertiary* actually hold in the *rôle* of syphilis; whether the more advanced of them are really due to the persistence of the disease and its still active presence in the system, or whether, with Mr. Hutchinson, we are to regard them as evidences of injury inflicted upon the economy by a disease which has now come to an end.

This latter view of the subject receives strong support from the circumstance that the more remote affections are, so far as we know, incapable of transmitting the disease from which they originated, either by contact, inoculation, or inheritance; and further confirmation is also afforded by some of these instances of reinfection, for no less than six of them occurred in persons who were suffering at the time from tertiary syphilis.[2] Unless, then, it be conceded

[1] Loc. cit., Obs. 6, 8, 9, pp. 8, 9, 10.

[2] DIDAY.—'De la Réinfection Syphilitique,' 1862, pp. 26, 27, Obs. xxix, xxx. BOULEY.—'Annales des Mal. de la Peau et de la Syphilis,' October, 1851; vol. iv, p. 9, Obs. ii. KÖBNER.—'American Journal of Syphilography and Dermatology,' April, 1873; vol. iv, p. 134, case viii. MERKEL.—Referred to by Köbner in above journal, p. 135. CASE IV related in this paper.

that two distinct attacks of general syphilis can coexist in the same individual, we must conclude that the so-called tertiary accidents which were present when the reinfection took place are merely the tokens of a bygone malady, and not the evidences of an existent disease.

But, whilst admitting that certain of the symptoms now classed as tertiary are merely the consequences of a lapsed syphilis, I cannot doubt that the earlier of them should be placed to the active operation of the syphilitic poison. It is true that they occur at a time when the economy has been subjected to the lowering influence of syphilis for a lengthened period, and possibly the peculiar forms they assume may be determined by the constitutional impairment, and so modified by it as to acquire the characteristics of those idiopathic affections which result from depraved health or defective vitality, however occasioned; but at first they so generally respond to the influence of special remedies that I am compelled to believe that they must be due to the continuous working of the disease in the system. A time, however, comes at length—marked by grave cachexia, unhealthy plastic deposits, and intractable ulcerations—when specific treatment ceases to be of service, and when tonics, liberal diet, and general care prove far more beneficial than special medicines; when this condition is reached the lesions attending it do not, I think, result from a still active syphilis, but from the degradation which the blood and tissues of the body have suffered in consequence of its previous effects.

I must apologise to the Society for the lengthy digressions in this communication, but the cases seemed to lead to the consideration of certain points still unsettled, the interest and importance of which must be my excuse for having introduced them.

TABLE *of Cases of Syphilitic Reinfection.*

Author.	No. of cases.	Reference.	Remarks.
Bergh[1]	2	Hospitals Tidende, Mar., 1865, Kopenhagen	Both patients had constitutional disease as the result of the reinfection.
Bjorken[1]	1	Upsala Läkareforningrs Fördhandlingen, iv, 7. pp. 597-624	The patient had general syphilitic symptoms from the second contagion.
Bouley	1	Annales des Maladies de la Peau et Syphilis, vol iv, p. 9, Obs. 2. Also related by Vidal, (de Cassis), Traité des Maladies Vénériennes	This was a case of successful inoculation from mucous tubercles in a woman suffering at the time from tertiary syphilis.
Boulongne	2	Recueil de Médecine, de Chirurgie, et de Pharmacie Militaires, 3e série, t. ii, p. 428, 1859. Also reported in the Gazette Médicale, 1859.	Both of these patients had indurated chancre and constitutional symptoms following the second infection.
Beauchet.....	1	Moniteur des Sciences Méd. et Pharm., Jan. 14, 1860. Also mentioned in Recueil de Méd., de Chir., et de Pharm. Militaires, 3e série, t. iii, p. 181, 1860	Indurated chancre and general syphilis resulted from the reinfection. Obs. xxiv of Diday's series.
Coote (Holmes)	1	Report of Committee appointed by the Admiralty to inquire into Pathology and Treatment of Venereal Diseases, 1867, p. 343	Constitutional symptoms followed the second infection.
Dardel.....	1	Gazette Médicale de Lyon, Août 16, 1865	Eruption followed the reinfection.
Delestre	1	Gazette Hebdomadaire, 1860, p. 56. Also reported by Follin, Traité de Pathologie Externe, t. i, p. 740, 1861, and Presse Médicale, 1860	Indurated chancre and constitutional disease resulted from the second contagion. The patient was seen by Ricord, Cullerier, and Puche; he had been under Ricord's care for chancre and eruption 20 years previously.

Author.	No. of cases.	Reference.	Remarks.
Diday	27	De la Réinfection Syphilitique, 1862, and also Archives Générales de Méd., Juillet et Août, 1862	Thirty cases are given by Diday, but Obs. 18 was reported by Rodet, Obs. 23 by Follin, and Obs. 24 by Beauchet, in whose practice they respectively occurred, and they are placed, therefore, under these authors. Of the remaining 27 cases, 14 had an indurated chancre only for second infection, 8 had constitutional disease as well, 2 were cases of indurated chancre in persons at the time under the influence of tertiary disease, and 3 had indurated chancre only for the first contagion.
Engelsted[1]	1	Bericht über das Commune Hospital in Kopenhagen, 1868, p. 115	This patient had constitutional syphilis.
Follin	1	Traité de Pathologie Externe, t. i, p. 740, 1861. Also reported in Gazette Hebdomadaire, 1854, t. i, p. 213; and referred to by Vidal (de Cassis), Traité des Mal. Vénér., 2e édit., p. 406	Had been in the Hôpital du Midi, under Puche, three years before, with indurated chancre and bi-inguinal adenopathy, but no other general symptoms. Indurated chancre and eruption for second infection. Obs. xxiii of Diday's series.
Hardie	1	Report of Admiralty Committee on Venereal Diseases, 1867, p. 162	Eruption followed the second contagion.
Hilton.....	1	Ditto, ditto, p. 399	Eruption resulted from the reinfection.
Huggenberger[1]	2	Zur Frage von der Einmaligkeit der Const. Syphilis. Petersburg Med. Ztschr., 1862, Bd. 3, p. 161	Both patients had general syphilis.

Author.	No. of cases.	Reference.	Remarks.
Hutchinson ...	2	Report of Admiralty Committee on Venereal Disease, 1867, p. 288. This case is referred to also in Reynolds' System of Medicine, vol. i, p. 293	Both these patients suffered from constitutional syphilis as the result of the second contagion.
		Lancet, 1874, vol. i, p. 157	This patient had three distinct attacks of syphilis.
Köbner	8	American Journal of Syphilography and Dermatology, April, 1873, p. 128, translated from the Berliner Klinische Wochenschrift for November, 1872	In six cases the second infection showed itself in the form of indurated chancre only; in the other two general syphilis was also present. One of the former cases occurred in a patient suffering from tertiary disease.
Lindwurm [1] ...	1	Würzberg, Med. Ztschr., Bd. 3, Hft. 3, p. 153	Constitutional symptoms resulted from the reinfection.
Merkel, Joh.[1] ...	1	Bair. Intellig. Bl., 1869, 22	Chancre and eruption occurred whilst the patient was suffering from syphilitic disease of bone.
Rodet	4	Union Médicale, Août, 1857, p. 425, et seq. The second case has been published in the Gaz. Méd. de Lyon, 1857; also by Diday, De la Réinfection de la Syphilis, 1862, Obs. xviii; and by Follin, Traité de Path. Ext., t. i, p. 740, 1861, Case 2	All these cases had constitutional disease following the reinfection.
Zeissl [1]	1	Syphilis Constitutionelle, p. 48	The second contagion was in the form of an indurated chancre only.

[1] For most of these cases and their references I am indebted to a paper in the 'American Journal of Syphilography and Dermatology' for April, 1873, "On Reinfection in Constitutional Syphilis," by Dr. Heinrich Köbner, translated from the 'Berliner Klinische Wochenschrift,' November, 1872.

In analysing these 60 cases it will be seen that in 35, constitutional symptoms followed the second contagion; in 20, indurated chancre alone was the result; in 5, an indurated chancre occurred whilst the patient was still suffering from tertiary disease; and in one of these (Merkel's case) a papular eruption also showed itself The large number of cases attended by general manifestations may, perhaps, be accounted for on the surmise that many practitioners who have met with an indurated chancre in subjects whom they knew to have had syphilis previously, have not placed them on record, thinking that they must have been mistaken as to the character of the sore, or that it had become hardened from some extraneous cause, and consequently have reported only those instances which were attended by constitutional symptoms. But this might lead to error as to the comparative frequency with which a second contagion is followed by general symptoms, for when several cases have been observed by the same individual it has been found that an indurated chancre only is the most common result of reinfection, as may be seen by reference to Diday's and Köbner's cases. My own series, though, gives a different return.

ON THE

LARYNGEAL SYMPTOMS

WHICH RESULT FROM THE

PRESSURE OF ANEURISMAL AND OTHER TUMOURS UPON THE VAGUS AND RECURRENT NERVES.

BY

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Amongst the practical gains which have resulted from the use of the laryngoscope one of the greatest has been the increased facility which the inspection of the living larynx has afforded for investigating the results of disease implicating the vagus and recurrent nerves.

The main object of the present communication is first to demonstrate and then to explain the fact that *bilateral spasm* and *bilateral palsy* of the intrinsic muscles of the larynx may result from the pressure of an aneurism or other tumour *on the vagus nerve of one side only*.

Two cases of bilateral palsy of the larynx with an aneurism pressing on one vagus and recurrent nerve have recently been recorded: one by Dr. Baümler, in the twenty-third volume of the 'Pathological Transactions;' the other by myself, in the twenty-fourth volume of the same Transactions.

My own case ('Path. Trans.,' xxiv, p. 42) was that of a man forty-five years of age, who for several months had been suffering from stridulous and difficult breathing, the voice being feeble but tolerably clear. Looking into his larynx with the mirror, I saw the vocal cords of their natural colour, nearly touching each other in the middle line, and nearly motionless. There was a slight approximation of the cords during vocalisation. During inspiration the glottis did not expand as in the normal state, but, on the contrary, the cords appeared to be pressed nearer together by the inspiratory current of air, while in expiration again the cords were slightly pushed apart by the outgoing stream of air. No swelling or other structural change within the larynx was visible. There was dulness on percussion over the *manubrium sterni*—an impulse was heard there at each systole of the heart, and an impulse was felt by the ends of the fingers when firmly pressed against the bone. The patient stated that eighteen months before he had suddenly become hoarse while talking to a friend; the feebleness of voice had continued, and the breathing had gradually become difficult and attended with a noise in the throat. For some months past the breathing had become so difficult, especially when lying down at night, that he had been unable to sleep for more than a few minutes at a time. I came to the conclusion that the intrinsic muscles of the larynx on both sides were paralysed by the pressure of an aneurism of the aorta on one or both recurrent nerves.

During the first night after his admission into the hospital he got no sleep, and several times he appeared to be on the verge of suffocation. The following day Sir William Fergusson, at my request, performed tracheotomy. The operation afforded immediate and great relief, and the next night the patient slept for several hours. Two days afterwards symptoms of pleuro-pneumonia set in, and he died on the fourth day after the operation. An aneurism about the size of an orange projected backwards from the transverse aorta. The left vagus nerve passed in front of the aneurism, and was closely involved in its wall; the left recurrent passed

round and behind the tumour, where it was compressed and atrophied, and nearly lost in the wall of the aneurism. There were some enlarged lymphatic glands near the right recurrent, but the nerve was not pressed upon or even touched by these glands, and both it and the right vagus appeared quite normal.

My friend and colleague Dr. Curnow did me the favour to dissect out the nerves and the muscles of the larynx, and he reported as follows :—"The laryngeal muscles on the left side are decidedly atrophied; those on the right side are somewhat larger, but I am inclined to think them atrophied also."

In Dr. Baümler's case ('Path. Trans.,' xxiii, p. 66) with the physical signs of aneurism of the innominate artery there was dyspnœa with loud laryngeal stridor and aphonia. The laryngoscope showed complete immobility of the right vocal cord, with immobility almost as complete of the left cord. After death, which occurred from increasing dyspnœa, the right vagus was found flattened, and in one place almost lost in the wall of an aneurism of the innominata; the recurrent branch was also flattened and thinner than that on the left side. The left vagus and recurrent appeared perfectly normal everywhere. The intrinsic muscles of the larynx were very pale and flabby, and there was no appreciable difference in the bulk of the muscles on the two sides. The microscope showed granular degeneration of the muscles on both sides to an almost equal extent.

Dr. Baümler mentions first amongst the points of interest in this case "the absence of changes in the left recurrent, although during life the corresponding muscles showed, on laryngoscopic examination, very great impairment of their function."

In both these cases of aneurism the fact of bilateral palsy of the larynx was ascertained by a careful and thorough laryngoscopic examination during life; in both cases the laryngeal obstruction was greater than an affection of the

[1] The preparation is in the museum at King's College.

muscles on one side only would account for. In both cases the post-mortem examination was made with the expectation of finding that the recurrent nerve on both sides had been injured, but in both instances it was found that the vagus and recurrent on *one* side only had been compressed; and, lastly, in both cases the intrinsic muscles of the larynx on both sides had undergone atrophic changes. In my own case the muscular atrophy was greater on the side of the compressed nerve, but in Dr. Baümler's case the wasting of the muscles appeared to be equal on the two sides.

For a long time the bilateral palsy of the larynx which unquestionably existed in these two cases appeared to me inexplicable, and I often discussed it with my physiological friends without obtaining a satisfactory explanation; but at length it occurred to me to suggest the following interpretation of the phenomena.[1]

The aneurism in each case compressed not only the recurrent branch, but also the trunk of the vagus; it seems, therefore, not improbable that while the muscles on one side of the larynx were paralysed by direct pressure on the recurrent branch on that side, the paralysis on the other side was the result of a centripetal irritation of the trunk of the vagus acting on the nervous centre, and through it upon the nerve-supply to the laryngeal muscles on the opposite side, so that the palsy on one side was direct, while that on the other side was the result of a reflex influence.

I now proceed to adduce in support of this theory such facts and arguments as I have been able to gather from clinical observation, and from the results of experiments on living animals.

It is an unquestionable fact that the intrinsic muscles of the larynx are in a pre-eminent degree bilateral in their action. Every one who has inspected the living larynx knows how absolutely impossible it is in the normal condition to move one vocal cord without at the same time moving the other to an equal extent.

[1] 'British Medical Journal,' June 27th, 1874.

According to Dr. Broadbent's ingenious and well-known hypothesis the muscles which thus act bilaterally must have their central nerve nuclei so closely connected by commissural fibres that the muscles of each side receive their nerve supply from both sides of the brain in proportion to the completeness of their bilateral action. The muscles of the larynx, which are in an especial degree bilateral in their action, in accordance with this hypothesis, must be on either side equally connected with both sides of the nervous centre. The central commissural connection between the nerves supplying the larynx is not a mere hypothesis or a physiological inference, but a demonstrated anatomical fact. Dr. Lockhart Clarke in his elaborate paper "On the Intimate Structure of the Brain"[1] has described and figured some of the fibres of origin of the spinal accessory nerve, decussating in three places across the median line of the medulla oblongata, and thus connecting the spinal accessory nuclei of the opposite sides. The spinal accessory nerve is known to be the source of the motor fibres in the laryngeal branches of the vagus, and in the structural arrangement thus demonstrated by Dr. Clarke, whereby the spinal accessory nuclei of the two sides are brought into close union with each other, we appear to have the explanation of the normal bilateral action of the laryngeal muscles. This commissural union of the nerve nuclei of bilaterally acting muscles explains certain well-known pathological phenomena.

1st. As Dr. Broadbent has shown, it explains the fact, that in cases of hemiplegia resulting from a lesion of one hemisphere of the brain, the muscles which act bilaterally are not paralysed. The reason is, that although the motor influence from one side of the brain to these muscles is cut off or lessened, the motor influence from the other side reaches the commissural centre, and thence passes to the muscles on both sides. Dr. Broadbent explains it thus: "If the centre of volitional action on one side is destroyed, or one channel of motor power is cut across, the other will transmit

[1] 'Philosophical Transactions,' 1868, Part I, pp. 272, 273.

an impulse to the motor centre, and this will be communicated to the nerves of the two sides; equally if the fusion of the two nuclei is complete, and there will be no paralysis; more or less imperfectly to the nerve of the affected side if the transverse connection between it and its fellow is not so perfect, in which case there will be a corresponding degree of paralysis."[1] The connection between the central origin of the nerves supplying the laryngeal muscles must be most intimate, for in cases of hemiplegia the result of a lesion of one cerebral hemisphere, however complete may be the palsy of the arm and leg, the muscles of the larynx appear to be quite unaffected by the paralysing influence. The voice, as a rule, is unchanged in these cases, and the laryngoscope shows an equally unimpeded movement of the cartilages and the vocal cords on both sides. In all cases of unilateral palsy of the laryngeal muscles the paralysing lesion must be below the junction of the nerve nuclei.

2nd. It is evident that the commissural union of the nerve nuclei, which explains the escape of bilaterally-acting muscles from *palsy* in cases of hemiplegia resulting from unilateral brain disease, renders these associated muscles liable to *spasm* when a morbid motor influence proceeds from either side of the nervous centre. Thus, in cases of hemispasm—spasm, that is, of one arm and leg starting from a lesion of one cerebral hemisphere—the bilaterally-acting muscles of the chest and abdomen on both sides are liable to be, and are in fact, spasmodically affected; so the epileptic cry which is sometimes heard at the commencement of a convulsive attack is a result and a proof of bilateral spasm of the larynx. In these cases the morbid motor influence reaches the commissural fibres, and thence passes to the nerves and muscles on both sides. Thus the same commissural union which protects bilaterally-acting muscles from the paralysing influence of a one-sided brain-lesion, renders them liable to be implicated in convulsive attacks resulting from a disease on either side of the brain.

3rd. It is obvious that if the nerve nuclei and the com-

[1] 'British and Foreign Medico-Chirurgical Review,' April, 1866.

missural fibres which connect them become diseased, bilateral palsy may result. And this is what probably happens as a result of a morbific influence conveyed to the nerve centres when the trunk of one vagus has been for a long time irritated by the pressure of an aneurism or other tumour.

Many cases are recorded, I have myself seen several, in which a tumour, aneurismal or cancerous, pressing on one recurrent nerve, has caused unilateral palsy of the intrinsic muscles of the larynx. The explanation of these cases is obvious. The conducting power of the efferent nerve is impaired or destroyed, and, as a necessary result, the muscles on that side are paralysed. It is equally obvious that pressure on one recurrent nerve alone, a nerve which is purely motor, and distributed only to the muscles on its own side, will not, either directly or indirectly, paralyse the muscles on the opposite side of the larynx. But it is, to say the least, extremely probable that pressure on the trunk of one vagus may, through an influence conveyed by its afferent fibres to the nerve centre produce bilateral *palsy*, as in the two cases whose histories I briefly gave at the commencement of this paper; while in other cases, to which I shall hereafter refer, bilateral *spasm* of the larynx may be excited through the same nervous channels.

It occurred to me, that some light might be thrown upon this subject by experiment; and my friend and former colleague, Professor Rutherford, did me the favour to perform some experiments on living rabbits, with results which I will now briefly describe. In all the experiments, the animal being fixed on its back, the larynx and trachea were exposed, and the anterior wall of the trachea just below the larynx was cut away, so that the glottis could readily be seen from below.

The superior laryngeal nerve was exposed and divided, and its central end was stimulated by a faradic current; the immediate result was a strong bilateral adduction of the vocal cords; in other words, the glottis was spasmodically closed. This experiment was performed several times on four rabbits, and with uniform results. After we had performed this

experiment, we found that the same results had before been obtained by other experimenters; in particular by Rosenthal,[1] and by Waller and Prevost.[2] The explanation is obvious—a stimulus is sent through the afferent laryngeal nerve to the centre, and thence is reflected by the efferent fibres of the two vagi through the recurrent branches to the muscles of the larynx on both sides. This experiment then affords a good illustration of the physiological mechanism of bilateral spasm resulting from unilateral irritation.

The principle is probably the same, whether a morbid motor influence passes downwards from one side of the brain, or upwards through the afferent fibres of one vagus to the common centre of both vagi.

When the recurrent nerve on one side was stimulated, the vocal cord on the same side was abducted, while the opposite cord was unaffected.

Another experiment consisted in cutting across the trunk of the right vagus between the superior and the recurrent laryngeal nerves, and then applying the electrical stimulus to the lower end; the result was abduction of the vocal cord on that side, while the cord on the opposite side remained motionless. Rosenthal found that this experiment performed on a cat caused unilateral adduction of the vocal cord.[3] Whether abduction or adduction result from the stimulation of the recurrent or the distal end of the vagus depends probably upon the relative vigour of the antagonistic intrinsic muscles of the larynx.

When the central end of the right superior laryngeal was stimulated after the trunk of the vagus on the same side had been divided, the vocal cord on the opposite (left) side was adducted by the reflex nervous current, while the cord on the same side remained motionless in consequence of the efferent fibres in the trunk of the right vagus being cut across.

When the central end of the divided vagus is stimulated,

[1] 'Die Athembewegungen und ihre Beziehungen zum Nervus Vagus, Berlin, 1862, pp. 224, 225.

[2] 'Archives de Physiologie,' tome iii, 1870, p. 196.

[3] Loc. cit., pp. 211, 212.

the result is not adduction of the opposite vocal cord, as when the superior laryngeal is irritated, but Rosenthal observed that the diaphragm and other inspiratory muscles are made to contract, and with this inspiratory action the vocal cord is abducted.[1] Waller and Prevost also obtained the same results from the excitation of the central end of the vagus.[2]

These experiments serve to establish two general principles:

1st. Any movement of the larynx which results from stimulation of the afferent fibres or branches of one vagus is bilateral, except that, of course, division of the trunk of the vagus will prevent the occurrence of the reflex action on that side.

2nd. Stimulation of the efferent fibres of one vagus—the recurrent branch or the distal end of the divided trunk—causes movement on the one side only of the larynx.

In the course of our experiments we were enabled to verify an observation made long since by Longet, namely, that after the intrinsic muscles of the larynx have been paralysed by the division of all four laryngeal nerves, the glottis is closed by the compressing action of the inferior and middle constrictors of the pharynx during the act of deglutition. When after all the laryngeal nerves are divided the action of the constrictors of the pharynx has been suspended by the division of their fibres on one side, the glottis remains motionless during attempts at deglutition. Electrical stimulation of the central end of the superior laryngeal nerve always excites the act of deglutition, as Waller and Prevost have especially pointed out. We observed distinctly, as a result of such stimulation, that the vocal cords moved inwards and rapidly closed the glottis before the slower act of deglutition had commenced; but in two animals Dr. Rutherford divided the constrictors of the pharynx before the experiments on the superior laryngeal nerve were commenced, the object of this procedure being

[1] Rosenthal, p. 214.

[2] Loc. cit., p. 196.

[3] 'Archives Générales de Médecine,' tome xii, 1841, p. 423.

[4] 'Archives de Physiologie,' tome iii, 1870, p. 185.

to enable us the better to distinguish the closure of the glottis by the constrictors of the pharynx during the act of deglutition from that which is caused by the intrinsic muscles of the larynx.

It is impossible by any experiment upon an animal to imitate all the influences which are in operation when an aneurism is gradually but constantly compressing the vagus and recurrent nerve in the human subject, but the results of the various experiments to which reference has been made give support and probability to the theory that a long-continued irritation of the trunk of one vagus may, through its afferent fibres, so disturb the common centre of the two vagi as to cause either bilateral spasm or bilateral palsy of the laryngeal muscles.

Reference may here be made to some well-known facts illustrating the influence of a peripheral irritation in exciting pathological changes in distant parts through a reflex nervous influence.

Cases of traumatic tetanus afford one good illustration of this. A foreign body in a wound may not only excite a general spasm of the muscles through an influence conveyed to the spinal cord and thence reflected through the efferent nerves to the muscles, but in fatal cases, as Dr. Lockhart Clarke has demonstrated, the spinal cord is found to have undergone extensive structural changes.

In the sixth volume of the 'Transactions of the Clinical Society' I have published a case in which a piece of flint beneath the skin in a partially healed wound on the cheek excited facial neuralgia with facial palsy on the same side and trismus. These results of a reflex nervous influence all passed away after the removal of the foreign body which had unquestionably been their exciting cause.

During the passage of a renal calculus through the ureter there is often pain in the testicle on the same side and sometimes there is inflammatory swelling of the painful part. I extract the following from Sir James Paget's 'Surgical Pathology:'—"Whoever has worked much with microscopes may have been conscious of some amount of inflammation of

the conjunctiva in consequence of over-work. Now, the stimulus exciting this inflammation has been directly applied to the retina alone, and I have often had a slightly inflamed left conjunctiva after long working with the right eye while the left eye has been all the time closed. I know not how such an inflammation of the conjunctiva can be explained, except on the supposition that the excited state of the optic nerve is transferred or communicated to the filaments of the nerves of the conjunctiva, generating in them such a state as interferes with their nutrition."

It is a well-known fact that a foreign body lodged in one eyeball may, by a disturbing influence transmitted through the nerves and the nervous centre, excite destructive disease in the other eye; and it is a common and a successful practice to extirpate the primarily injured eyeball to prevent the induction of a secondary and sympathetic disease in its fellow.

The more intimate the nervous connection between two parts the greater is the probability that disease in one may excite sympathetic disorder in the other; the probability, then, is very great that irritation of the trunk of one vagus will gradually excite functional disorder, and even structural change, in the nervous centre and in the associated nerve on the opposite side.

Clinical observation has established the fact that laryngeal symptoms of some kind occur in a large proportion of cases of aneurism of the transverse aorta. Dr. Sibson has shown, in his elaborate analysis of the symptoms of aneurism of the aorta, that in cases of aneurism affecting the transverse portion of the arch the voice or cough was raucous or whispering, or inspiration was stridulous in 47·5 per cent.[1]

In the history of cases of aneurism symptoms are commonly attributed to pressure on the recurrent nerve, which could not possibly result from the implication of that nerve alone.

The recurrent, be it remembered, is an efferent motor nerve. When galvanized, as we saw, muscular contraction

[1] 'Medical Anatomy,' Fasciculus 5.

occurs only on the one side; no reflex bilateral contraction occurs, as when the afferent superior laryngeal nerve is stimulated. Pressure on one recurrent nerve may cause either unilateral spasm or unilateral palsy of the laryngeal muscles. Spasm from irritation of one recurrent would be indicated, as our experiments have shown, by abduction of the vocal cord from the median line, while a paralysed vocal cord remains motionless, or nearly so, in the median line during the act of inspiration. A unilateral palsy of the glottis may render the voice somewhat feeble, and it may cause some stridor on a deep inspiration, but it does not narrow the opening of the glottis sufficiently to seriously impede the breathing. I have seen a considerable number of cases in which the laryngoscope has shown unilateral palsy of the larynx, but in not one has distress of breathing been occasioned thereby. Dr. Bristow, in an interesting paper in the 'St. Thomas's Hospital Reports' (New Series, vol. iii, p. 205), refers to two cases in which unilateral palsy of the larynx was caused by the implication of one recurrent nerve in a cancerous tumour; but he states that in neither case "was there a trace of dyspnœa, either persistent or paroxysmal." The loud laryngeal stridor and the urgent dyspnœa which so frequently result from aneurisms and other tumours within the chest may be due to one of three conditions—1st, bilateral spasm of the laryngeal muscles, the result of irritation of one vagus; 2nd, bilateral palsy of the larynx, probably a later result of a tumour pressing on one vagus and recurrent; 3rd, palsy on one side, a direct result of pressure on the recurrent with spasm of the muscles on the opposite side, a reflex result of pressure on the trunk of the vagus.

When the dyspnœa and stridor are paroxysmal, coming on and going off with equal rapidity, the immediate cause is probably bilateral spasm of the larynx, and these attacks bear a striking resemblance to cases of laryngismus stridulus in children. On the other hand, long-continued laryngeal stridor and dyspnœa are commonly caused by *paralysis* of both vocal cords. The laryngoscope affords most valuable aid in the diagnosis of these interesting cases.

It is probable that violent and prolonged spasm of the laryngeal muscles may sometimes be directly followed by a paralytic condition, during which the stridor and dyspnœa continue, and death at length occurs from suffocation. This sequence of pathological events would seem to be analogous to the palsy which follows a fit of epilepsy, when the convulsed limbs are left paralysed. Dr. Hughlings Jackson ingeniously suggests that the violent physiological discharge of nerve-force through the motor fibres during the epileptic paroxysm may, for a time, suspend their functional activity, and thus cause the so-called "epileptic hemiplegia."

There is reason to believe that a bilateral affection of the larynx, either spasmodic or paralytic, is a very common result of pressure on the trunk of one vagus. The records of medicine abound with histories of cases in which death has resulted from laryngeal obstruction, and in which a post-mortem examination has shown a tumour pressing on one vagus or on the vagus and its recurrent branch. I know of no case in which death has resulted from pressure on *one recurrent* nerve alone; but Dr. John Reid states that he has in his possession "a preparation procured from the body of a young man who died very suddenly, with all the symptoms of suffocation, when seated with some companions round a fire, who were chatting and laughing. *Both* recurrents are imbedded in a firm yellowish tumour, through which they cannot be traced."[1]

It is not to be supposed that a unilateral muscular affection of the larynx could cause suffocation. In all the fatal cases of this kind there must have been either a bilateral spasm or a bilateral palsy of the laryngeal muscles. The following cases may serve as illustrations of the principle for which I am here contending.

In the year 1847 I was asked by a medical friend to examine for him the body of a man, aged 29, who died after a six weeks' illness, from what was believed to have been laryngitis. The symptoms had been cough with scanty expectoration, hoarseness, noisy and difficult breathing, coming on

[1] 'Physiological, Anatomical, and Pathological Researches,' p. 275.

in paroxysms, in one of which he died, notwithstanding an unsuccessful attempt to perform tracheotomy. This case occurred in the dark ages before the introduction of the laryngoscope, and the two experienced gentlemen who had attended the patient, one of them a general practitioner, the other a consulting surgeon, had no doubt that the case was one of laryngitis. We found after death a perfectly healthy larynx, but a cancerous tumour filled the concavity of the arch of the aorta and implicated the left vagus and recurrent. The right vagus and its branches were intact. In this pathological history we have the results of an experiment upon the vagus, and it can scarcely be doubted that the channel of nervous influence through which the cancerous tumour excited the fatal bilateral spasm of the larynx was the afferent fibres of the left vagus to the common centre, and thence through the efferent nerves to the muscles of the larynx on both sides.

In the 15th volume of the 'Pathological Transactions,' p. 72, I have published the case of a man, aged 31, who was admitted into the hospital with symptoms which were at first supposed to indicate laryngitis. There were hoarseness, cough, dyspnœa, and laryngeal stridor. When I first saw him, two days after his admission, the dyspnœa and stridor had ceased, and I clearly saw, with the aid of the mirror, a healthy larynx with free movement of its cartilages and cords. The laryngeal symptoms which had been present, and a difficulty in swallowing solids, excited a suspicion of aneurism, although a careful examination detected no positive evidence of such disease. Two days later death occurred from hæmorrhage. An aneurism of the transverse aorta had compressed the left vagus and recurrent, and had opened into the œsophagus. In this case the pressure on the recurrent had not been sufficient to paralyse the muscles which it supplied, and it is believed that the laryngeal symptoms which had existed at the time of his admission were the result of bilateral spasm excited by the pressure of the aneurism on the trunk of the left vagus.[1]

[1] The preparation is in the museum at King's College.

On the 23rd of September in the present year I saw, a few miles out of town, a gentleman, aged 40, who, for two months, had suffered from cough and dyspnœa, with stridulous inspiration. The superficial veins over both sides of the chest were enlarged, and the veins at the lower part of the neck were distended. There was dulness on percussion over the manubrium sterni, and a distinct systolic blowing was heard there. The stridor, on inspiration, was heard more distinctly on applying the stethoscope over the larynx than lower down over the trachea, and at the back over the spinous processes of the vertebræ it was more distinct in the middle cervical region, on a level with the larynx, than lower down over the upper dorsal spines opposite the arch of the aorta. The stridor, therefore, was the result of obstruction in the larynx, and not due to direct pressure on the trachea, such as occurred in a case of aortic aneurism which I once saw with my friend, Dr. Richards, of Winchester, and which I published in the 'British Medical Journal,' December 23rd, 1871, p. 720.

On proceeding to examine the larynx with the mirror, I found the fauces unusually sensitive, and not getting a complete view at once I discontinued the examination, expecting in a few days to have an opportunity to complete the investigation. Unfortunately, no such opportunity occurred. Two days after my visit the patient, who had been quietly talking to his wife, suddenly exclaimed, "I am being suffocated, help me." The breathing became more noisy and very difficult, the face was livid, the skin was soon bathed in a cold sweat, and in his agony he tossed about his arms and rolled on the floor. A neighbouring medical man, who was called in, gave him ether vapour, which appeared to relieve the extreme restlessness, but the breathing gradually became slower, consciousness was lost, and death occurred in about two hours after the sudden onset of the suffocating dyspnœa. It is a remarkable fact that until he lost consciousness he was able to speak distinctly.

I regret extremely that I did not hear of the death until it was too late to make a post-mortem examination. In the

absence of such an inspection some points of interest which might have been ascertained must remain doubtful.

I inferred from the symptoms and physical signs which I have described that an aneurism of the transverse aorta pressed on the superior cava, thus causing the venous fulness and on the left vagus causing the laryngeal symptoms. As the laryngeal stridor and dyspnœa had continued with little change or variation for two months, it seemed probable that bilateral palsy of the muscles of the larynx was the cause of the obstructed breathing. Whether the final struggle, which began so suddenly, and ended so rapidly, was a result of a rapid increase of the laryngeal palsy or of a sudden spasm affecting the partially paralysed muscles, it is not possible now to decide.

Whatever may have been the immediate cause of the obstructed breathing, there can be no reasonable doubt that the seat of it was the larynx and not the trachea. It is probable, therefore, that the prompt performance ef tracheotomy might have prolonged life for a time.

This case, in some of its features, is very similar to the celebrated case of the Earl St. Maur, of which so interesting an account has been published by our President.

With reference to the question of tracheotomy in cases of aortic aneurism, obviously, the main point is to distinguish between laryngeal obstruction, the result of spasm or palsy caused by pressure on the vagus, and narrowing of the trachea by the direct pressure of an aneurism on the anterior wall of that tube. In the former class of cases tracheotomy may afford great temporary relief, as in the case recorded at the commencement of this paper, while in cases of obstruction of the trachea near its bifurcation, the operation would be worse than useless.

One fact which we observed in the course of our experiments is worthy of note on account of its bearing upon the therapeutics of laryngeal spasm. We found on two occasions that when a rabbit was under the influence of chloroform, the electric stimulus applied to the central end of the divided superior laryngeal nerve did not excite spasmodic

closure of the glottis, although the slight respiratory movement of the cords continued. After the effect of the chloroform had passed away the same electrical stimulus caused the usual bilateral spasm of the glottis. Waller and Prevost also found that anæsthesia by ether or chloroform arrested the reflex movements which are ordinarily excited by the electrical stimulus applied to the central end of the superior laryngeal nerve in the cat.[1] This antispasmodic effect of chloroform vapour is similar to the undoubted influence which the chloral hydrate exerts in preventing or lessening laryngeal spasm, whether in the purely spasmodic attacks of laryngismus stridulus or in cases of laryngitis with spasmodic complication, such as occurs very frequently in children and sometimes also in adults of both sexes.

The main points which I have endeavoured to establish in this communication are the following:

1. When serious laryngeal dyspnœa results from the pressure of a tumour on the vagus and its branches, the immediate cause is a bilateral affection of the muscles of the larynx. There may be bilateral spasm or bilateral palsy or palsy on one side with temporary spasm of the muscles on the opposite side.

2. Pressure on one recurrent nerve can affect the laryngeal muscles on the one side only.

3. Pressure on the trunk of the vagus may through an influence upon the nervous centre cause either bilateral spasm or bilateral palsy of the larynx, and this is the true physiological interpretation of the urgent laryngeal symptoms which are often excited by the pressure of aneurismal and other tumours upon the vagus.

4. The bilateral spasm of the larynx which is excited by the pressure of a tumour on the trunk of one vagus is, probably, the result of a morbidly exalted excitability of the nervous centre induced by irritation of the afferent fibres of that nerve, and in consequence of this state of exalted sensibility ordinary stimuli, acting on the afferent nerves of the larynx, may cause bilateral spasm similar to that which is

[1] 'Archives de Physiologie,' tome iii, 1870, pp. 188, 189.

excited by the extraordinary stimulus of electricity applied to the central end of the divided superior laryngeal nerve.

5. It is probable that the long-continued irritation of the trunk of the vagus may gradually, as in cases of traumatic tetanus, induce such demonstrable structural changes in the nerve-centre as will explain the bilateral palsy which appears to be one of the results of this chronic nerve-irritation.

In any future cases of this kind that may occur and prove fatal after the laryngoscope has shown the existence of a bilateral palsy of the larynx, it will be desirable to subject the medulla oblongata and the nuclei of the spinal accessory and vagi nerves to a careful microscopic scrutiny. If the result of such an examination should be the discovery of structural changes in the nervous tissue the evidence in support of my theory will be complete.

Postscript.—Dr. Weir Mitchell, of Philadelphia, has recently done me the favour to send me a copy of a paper published by himself in conjunction with Dr. Morehouse.[1] In this paper the authors describe a peculiar arrangement and distribution of the laryngeal nerves in the turtle, which appear to have an especial interest in relation to the subject of the preceding communication. In the turtle the movements of the glottis are effected by two pairs of muscles; by one pair of muscles the glottis is opened, while by another pair it is closed. The glottic muscles are supplied by two pairs of nerves, a superior laryngeal, and an inferior laryngeal or recurrent. The superior laryngeal nerves supply both the opening and the closing muscles of the glottis, while the inferior or recurrent laryngeal are distributed only to the opening muscles of the glottis. The superior laryngeal nerve, in addition to being the chief motor nerve of the larynx, in this respect being analogous to the recurrent in the higher animals, is proved by experiment to be the sensitive nerve of the larynx in the turtle. But the remarkable

[1] "Researches upon the Anatomy and Physiology of Respiration in the Chelonia," 'Smithsonian Contributions to Knowledge,' Washington, 1863.

arrangement which Drs. Mitchell and Morehouse have discovered consists in the existence of a true *chiasm* or decussation of fibres between the two superior laryngeal nerves, as a result of which each superior laryngeal nerve contains fibres from both sides of the nervous centre. This nervous chiasm resembles that of the optic nerves, and is, the authors believe, the only example of this arrangement yet observed in extracranial nerves. The authors were led to the discovery of this inter-communication between the superior laryngeal nerves by the results of experiment. They found that after both of the inferior laryngeal and one of the superior laryngeal nerves had been divided, the movements of the glottis continued unimpaired. They also found that Faradisation of the distal end of the divided superior nerve caused bilateral movement of the glottis. This result suggested the existence of some connection between the nerves of opposite sides. The inter-communicating fibres were then sought for and discovered in front of the larynx. It was found that after dividing the chiasm, division of one superior laryngeal nerve caused palsy of the glottic muscles on the same side. It thus appears that in the turtle the bilateral action of the laryngeal muscles is effected or assisted by such an inter-communication between the two principal motor nerves as gives to the muscles on either side an equal nerve-supply from both sides of the nervous centre. In the higher animals this union between the motor nerves of the larynx exists at their centre of origin.

Drs. Mitchell and Morehouse observed that after division of both inferior laryngeal nerves electric stimulation of the central end of one divided superior laryngeal nerve caused a reflex bilateral movement of the glottis. This shows that in the turtle, as in the higher animals, there is a central connection between the afferent fibres on one side and the efferent fibres on the other.

The correctness of the authors' conclusions as to the function of the laryngeal chiasm was confirmed by an experiment on one animal, which gave a result at first sight contradictory.

In a large turtle, the two inferior laryngeal nerves having been divided, division of the right superior laryngeal caused palsy of the glottis on that side. This result was different from that previously obtained by the like experiment, and on a careful dissection it was found that the interlateral communicating fibres (the chiasm) were involved in a mass of diseased tissue, which had the same effect as division of the chiasm had been found to have in previous experiments. This observation, which at first appeared to throw doubt upon those which had preceded it, did, in fact, prove the correctness of the authors' previous conclusions.

NOTES

ON

AN EPIDEMIC OF

MALARIOUS YELLOW FEVER

OCCURRING ON BOARD H.M.S. DORIS SHORTLY AFTER LEAVING PORT ROYAL, JAMAICA, MAY 18TH, 1873.

BY

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WRITERS distinguish between "malarious" yellow fever and what they term "specific" yellow fever. The former they classify as a remittent, the latter as a continued form of fever. They also state that albuminous urine, and suppression of urine, occur in the specific disease, but not in the malarious form of the fever. This, I find, is all the difference that they acknowledge as existing between the two. I must here state that the detached squadron, consisting of six frigates, arrived at Port Royal, Jamaica, on May 14th, 1873; here the Doris remained at anchor till May 21st, when she steamed up to Kingston, and gave leave to the crew. The Doris remained at Kingston till the 27th May, the men having had free communication with the shore the whole time. On the 28th May the squadron left Jamaica for the island of Nassau, New Providence. Port

Royal and Kingston at the time of the visit of the detached squadron were reported to be quite healthy and free from yellow fever. The temperature of the air was, however, high, viz. 84° to 85° Fahr., and it remained so night and day; at the same time, as it was nearly always a calm, the air felt close and stuffy; even the regular sea-breeze sometimes forgetting to pay its accustomed morning visit. After 5 o'clock in the evening the air was still, not a breath was stirring, the nights were hot and stuffy, and the malarious odours from the shore could be distinctly smelt on board the ship, which was from 50 to 100 yards from the shore.

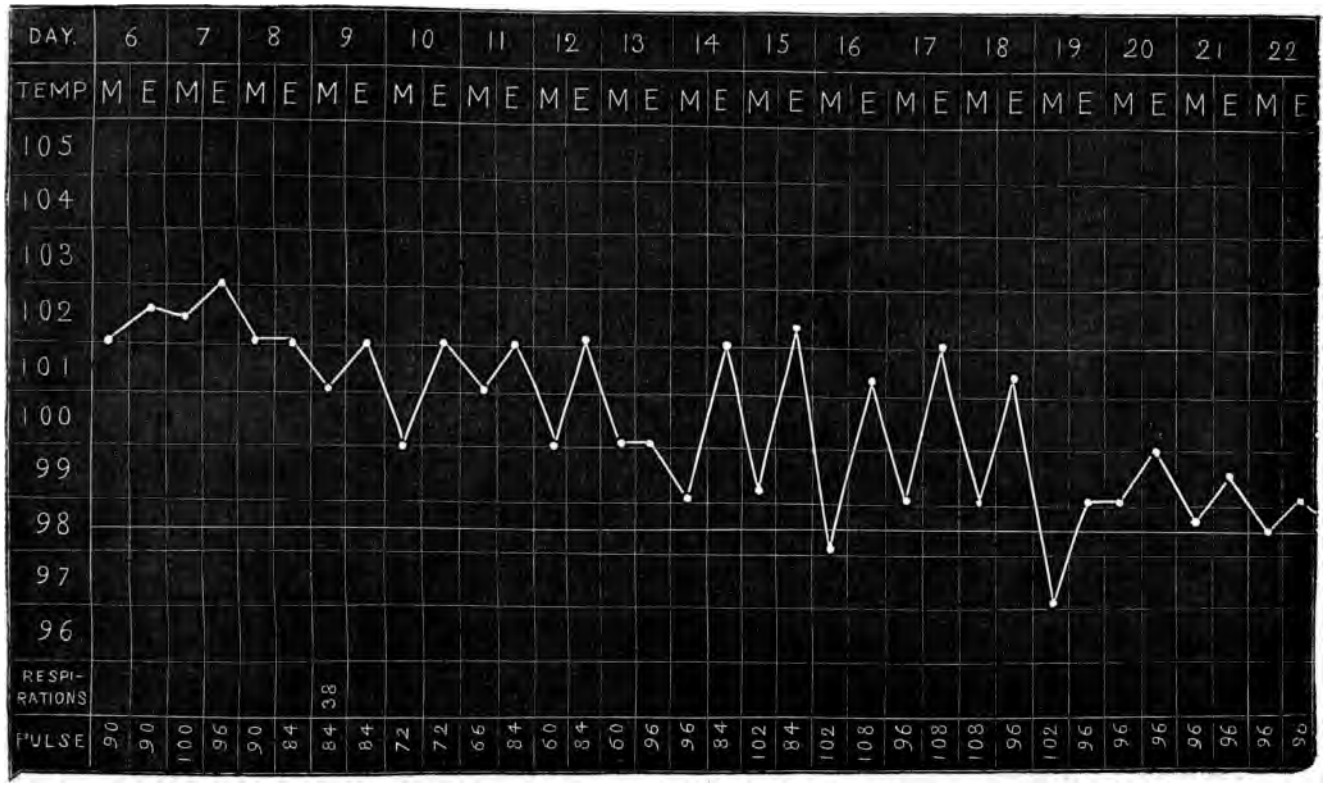
Between May and October is the malarious yellow fever season, so fatal to visitors to the island of Jamaica, whose seaports are situated low down on the sea-shore, the land being flat and swampy.

Most authors agree that it is most difficult to distinguish between malarious yellow fever and the so-called specific yellow fever.

The first case of malarious yellow fever happened on board the Doris on May 28th, the day the ship left Port Royal for Nassau. This was the case of Thomas Y—, whose temperature chart it will be seen has much the appearance of a case of typhoid fever in its later stages, but this man never suffered from any typhoid symptoms; he was evidently suffering from a malarious fever; he complained of headache, giddiness, epigastric pain, nausea, loss of appetite, and muscular debility; his skin was hot, face sallow and pinched, tongue coated with a creamy fur, urine scanty and high-coloured, but not albuminous, of normal specific gravity; the temperature in the axilla but slightly above the normal at first, in the course of a few days rose to 103° Fahr., and continued high until the sixteenth day of the fever, rising at night and falling in the morning with great regularity.

He was given an emetic in the first instance, afterwards saline mixture, then quinine. He had bland diet, port wine, iced drinks, and barley water. Although he suffered from nausea he never had much vomiting, and was convalescent by the 20th day; his temperature, however, was still above

Remittent fever (malarious yellow fever). Temperature Chart.—Case of Thomas Y—, æt. 24, A.B.



Line of Normal Temperature, 98·4°. M. Morning; E. Evening.

the normal, and the sallowness and pinched appearance of the face remained, and he still occasionally suffered from giddiness. Had there been no evidence of an epidemic of malarious fever existing at this time I should have considered this a case of a continued type of fever, for there was never any genuine remission of the symptoms.

The day after this case happened two other cases occurred. One of these, Thomas N—, was so similar to the one above described that it would be a mere repetition to detail it here.

The other of these two cases, however, Thomas M—, suffered from the same symptoms as did the two previous ones for the first three days, when the fever became more sthenic in its nature, and he died on the fourth day of undoubted black vomit. He was a fine healthy and muscular young fellow, of very temperate habits generally, but on this occasion appears to have broken out into excesses at Kingston after the violent exertion of boat racing. He was placed on the sick list on May 29th, 1873, with febrile symptoms, having been exposed to malarious influences on May 24th. On this day he was pulling in a racing boat at Port Royal, and in the evening he went on shore at Kingston to enjoy himself; he was picked up in a gutter intoxicated that night, and came off to the ship next morning; he went on shore again, I believe, several times during the next two or three days. On the morning of the fourth day of the fever, there having been nothing remarkable in his previous symptoms, the temperature in the axilla rose to 103° Fahr.; he now had much nausea and vomiting of frothy mucus; the skin and conjunctivæ were yellow, and he had pain in the epigastric region. He was ordered a sinapism over the stomach, saline mixture, bland diet, iced lemonade, barley water, and wine. At noon he said he felt better, sat up in bed and took his dinner, but I noticed at this time that his eyes had a dark rim round them, and the lids were puffy. About 7 o'clock in the evening he suddenly became unconscious, and had stertorous breathing, blowing respiration, and great oppression in the præcordial region, as evidenced by

palpitation and laboured breathing; the respirations were 34 per minute; pulse 120, compressible; temperature 104° Fahr. in the axilla; pupils dilated. The head was shaved, a blister applied to the nape, iced water on lint to the head, and ice was applied over the heart, which at once moderated its action and improved the pulse, which had become very feeble. A turpentine enema was administered, which brought away a large, black, fluid evacuation, very fœtid, and which continued to flow from him at intervals for some half hour. Mustard plasters were applied to the calves of the legs; an attempt to give stimulants by the mouth failed, as he could not swallow. At 8.30 p.m. some frothy mucus tinged with blood oozed from the angles of the mouth, and a few minutes afterwards the characteristic black vomit appeared, being ejected with much violence, to the amount of about a pint. Turpentine stupes were now applied over the stomach; the pulse became feebler, and a peculiar odour was given off from the body and lungs. At 10.25 p.m. violent vomiting again commenced, the vomited matter was quite black, like black and clear coffee, and he was now bathed in a cold clammy perspiration, with a rapidly failing pulse. He died strongly convulsed in the course of the next five minutes, viz. at 10.30 p.m., just three hours and a half after the graver symptoms had commenced, and on the fourth day of the fever. This case towards its termination had very much the appearance of a case of heat apoplexy, such as I have often witnessed in the Red Sea, until the black vomit appeared, and I have never seen black vomit in heat apoplexy; some authors consider it a form of remittent fever. This man had passed no water since the morning, and the bladder seemed empty at death; his urine had been free from albumen previously. No post-mortem examination was allowed to be made in this case, as the ship was in the tropics, and it was thought there might be a danger of spreading the infection, or, if the disease did spread afterwards, it might be put down to the "sectio cadaveris." But I examined the vomit; Nessler's test showed ammonia to be present. The reaction was slightly acid. Magnified

600 diameters there were seen: epithelium scales broken up; white corpuscles which were much in excess of the red blood corpuscles, in the proportion of about four to one; the red ones were not arranged in rouleaux, but were either coalesced irregularly, or distributed as single corpuscles. The colourless bodies were of two sizes, the smaller bodies being evidently free nuclei, becoming disengaged from broken-up corpuscles; the red corpuscles seemed to be more generally broken up and decomposed than the white ones. There were also seen a number of bodies very closely resembling spermatozoa or microscopic worms, but there was no motion in them at the time of examination, twelve hours after death. There were no vibriones or other forms of either animal or vegetable life present in the vomit. As this was the only case during the epidemic that suffered from genuine black vomit I had no further opportunity of examining it.

Between May 29th and June 9th eight fresh cases of fever occurred, with similar symptoms to the previous ones; four of these, being mild cases, were sent to duty after from two to thirteen days' treatment; the other four cases were, however, of a much graver description; three of these suffered from violent retching and bilious vomiting; the vomited matter, at first white or yellow, became afterwards very dark green —almost black. The vomited matters were all of slightly acid reaction.

Seeing the turn matters were taking I thought it advisable to recommend that the ship should go at once to sea and proceed northwards, so as to get a temperature of 60° or 50° Fahr. if possible, the temperature of the air where we then were at the island of Nassau being 84° Fahr. The Doris was at once ordered to proceed to Halifax, and left for that northern port the same day. Directly we got well out to sea, with a fresh and cool breeze blowing, the fever cases began to improve, the vomiting became less urgent and gradually stopped, remedies were of greater service, and the temperature of the air lowered.

Up to June 13th, however, eleven fresh cases happened

while we were at sea; they were, however, milder than the previous ones, six of them being sent to duty after three or four days' treatment. After this date no fresh cases occurred, and when the ship arrived at Halifax on June 18th only nine cases remained under treatment, and these were all convalescing, the temperature in the axilla in some still rising every night to 100° and 101° Fahr. They were sent to hospital at Halifax for further treatment, as it was considered advisable to get them out of the ship.

We had had a gale of wind blowing us along for several days past, and the air temperature had fallen to 49° Fahr. This is a temperature in which it is simply impossible for yellow fever, whether malarious or specific, to exist.

There were twenty-two cases in all during this outbreak, and only one death.

The following is a statement of the dates of the occurrence and duration of the attacks, with the results of treatment:

TABLE *of Cases of Malarious Yellow Fever and Remittent Fever occurring on board H.M.S. Doris,* 1873.

Date of exposure to malarious influences.	Date of attack.	No. of cases.	Convalescent and sent to hospital.	Well, sent to duty.	Died.	No. of days under treatment.
From May 14th to May 28th, 1873, at Port Royal and Kingston, Jamaica.	1873					
	May 28	1	1	...	...	22
	„ 29	1	...	...	1	4
	„ „	1	1	...	...	21
	June 2	1	...	1	...	13
	„ „	1	...	1	...	5
	„ 4	1	...	1	...	2
	„ „	1	1	...	...	15
	„ 6	1	1	...	...	13
	„ 7	1	...	1	...	8
	„ 8	1	1	...	...	11
	„ 9	1	1	...	...	10
	„ 11	1	1	...	...	8
	„ „	1	...	1	...	4
	„ 12	1	1	...	...	7
	„ „	1	...	1	...	3
	„ „	1	...	1	...	3
	„ „	1	...	1	...	3
	„ „	1	...	1	...	3
	„ „	1	...	1	...	3
	„ 13	1	1	...	...	6
	„ „	1	...	1	...	5
	„ „	1	...	1	...	5
	Totals.....	22	9	12	1	

With regard to the temperature of the cases of malarious yellow fever that came under my observation during this epidemic, in the milder cases the temperature was raised 1° to 2°, but in the more severe as much as 6° or 7° above the normal. It was by no means invariably higher at night than in the morning.

In the case of C. J. R— the temperature nearly every night was 101° to 103°, falling every morning to 99° or 98°, after the first five days, up to the sixteenth day, when he was sent to hospital.

But in most of the cases the temperature was higher in the morning than it was in the evening; this was more especially the case during the first seven days.

In the case of James S— the evening temperature was 96° to 98·4°, the morning 103° to 104°, for the first six days, after this it became normal.

Daniel McT— had a temperature of 103° on the first morning, it was normal the same evening; the next morning it was up as high as 105°, and down to 100° in the evening of the same day; on the third day it was 104·2° in the morning and 98·4° in the evening; on the fourth day the morning temperature was 103·8°, the evening 98°; from the sixth to the eleventh, when he was sent to hospital, the morning and evening temperatures fell a little below normal.

I consider this epidemic to have been of the malarious yellow fever type, that is to say, a remittent form of fever; and although only one case actually had black vomit, some of the others were bordering very closely upon it. The patients when first attacked were generally seized somewhat suddenly with fainting, præcordial oppression, hurried breathing, epigastric pain, giddiness, and nausea, the temperature being from 1° to 3° Fahr. above the normal, and in some of the more severe cases as much as 6° or 7° Fahr. above the normal. A hot stage followed, and in some cases lasted two or three days without the least sign of a remission.

Before we left Nassau for the north the cases were of the sthenic form, but after we got to sea the fresh cases were decidedly asthenic in their nature, the last cases being much milder than the preceding ones; nevertheless, they had all of them giddiness, muscular debility, and epigastric pain, with about 2° elevation of temperature, and a feeble pulse.

The urine was neither suppressed, nor was it albuminous in any of the cases, nor did it contain blood; it was somewhat scanty and high coloured; the specific gravity normal.

The fever was of the continued type, the first three or four days, afterwards in some cases it assumed the quotidian type, in others the tertian, and in others, again, an irregular remittent; a reference to the temperature chart (p. 51) will show this.

"There are many grades of intensity in remittent fever

(varying as it does from a severe intermittent to malarious yellow fever)."—*Aitken.*

Some of the cases were attacked in the morning and others in the evening. The period of convalescence required, and received, close attention with regard to diet, &c; and the speedy and complete removal from malarious influences, together with a much colder climate, I feel certain operated favourably in enabling the system the better to assimilate food, &c., and the sooner to eliminate the malarious poison; perhaps cutting short the attack of fever in some cases, and most certainly assisting materially the stage of convalescence.

The treatment generally pursued was this—in the first instance an emetic was given, afterwards a purge; on the following day, if the bowels were confined, a turpentine enema was administered, and turpentine was given by the mouth; sometimes a blue pill was ordered; sinapisms were applied to the epigastrium, the hair was cut close or shaved, and ice was applied to the head. After a few days quinine in full doses was invariably given. The diet was of the blandest until convalescence was established, when the patients were allowed some chicken, puddings, &c. A liberal allowance of port wine formed part of the diet. For drink they had iced lemonade, barley water, and iced tea, and the cold tea was much better tolerated than any other fluid, where vomiting was at all troublesome, and it was preferred by the sick. Acid drinks were neither tolerated well nor liked.

Aitken says, "When an observer has seen only the milder form of 'marsh remittent fever,' and is then suddenly called upon to witness an attack of 'malarious yellow fever,' he may well believe that the affections are entirely distinct." And, again, the same authority says, "The diagnosis between the 'specific contagious yellow fever' and the malarious form of yellow fever is at all times difficult; and severe 'marsh fevers' in certain geographical limits have a close resemblance to contagious yellow fever."

ON THE PATHOLOGY

OF

LUPUS ERYTHEMATOSUS.

BY

GEORGE THIN, M.D.

(Received November 20th, 1874—Read January 12th, 1875.)

THE diseased condition of the skin described by Hebra as *Seborrhœa congestiva* in 1845, and by Cazenave as *Lupus erythematosus* in 1853, had not escaped the notice of previous observers. But to Hebra belongs the merit of having first given such a clear description of the distinctive appearances, as permits of its being easily separated from other diseases with which it is liable to be confounded. His adoption of Cazenave's designation, although doubtless not made without sufficient reason, has nevertheless had the inconvenience of causing a frequent confusion of this disease with certain forms of lupus vulgaris from which it is specifically distinct.

For a detailed account of the malady I refer to Neumann's 'Lehrbuch,' and to the exhaustive article by Kaposi, in Hebra's 'Textbook of Skin Diseases.' I shall here give only such a short sketch of its symptoms and course as is

necessary to show their connection with the pathology with which this paper is more immediately concerned.

Small red spots which do not completely disappear under pressure by the finger, and which by their junction form erythematous patches having a circular border and of various sizes, constitute the primary feature of the disease. The patches so formed have a slightly raised reddened edge, and in the centre are either covered with greasy closely adhering scales or have a sunken puckered cicatricial appearance. On the face, which is by far its most frequent seat, and especially on the nose, the scaly masses when removed present the peculiarity of having on their adherent surface numerous projecting processes, which are moulds of the widened mouths of the sebaceous follicles, now seen interspersed over the congested surface.

The disease may last for months or years, sometimes making little progress, sometimes healing at one part and advancing at another, the skin after its disappearance appearing either normal or having the cicatricial aspect to which I have referred. It may defy all treatment, while the general health of the patient may at the same time be excellent. The affected spots on the scalp which are present in most instances become bald.

In the majority of cases there is little more to be described, the essential characteristics being, in fact, fairly included in three points by Cazenave, namely, in redness, wasting of the skin without ulceration, and the identity of its nature under different aspects.

It differs essentially from lupus vulgaris in the absence of ulceration, in its affecting people who are in the prime of life, rarely beginning until after the age of twenty, and in its characteristic general appearance.

There remains, however, a minority of cases in which another chapter must be added to complete the history. After the disease has lasted in the above-described chronic state for an indefinite time, it appears in the form of acute eruptions, and not only on the face, but simultaneously on many parts of the whole body. Hundreds of points may

spring up on the trunk and extremities in a few days. Kaposi records three cases in which the eruption appeared suddenly in the form of hæmorrhagic vesicles like crops of herpes. The complications of these severer forms of the disease are erysipelas, adenitis, anemia, and fever; its termination being frequently fatal to life.

The nature of the morbid processes in the skin which determine this singular group of symptoms is naturally a subject that has excited deep interest amongst dermatologists, and the investigations of Neumann, Geddings, and Kaposi on this point are of the first importance. Hebra, reasoning from the appearance of the distended and plugged ducts of the sebaceous follicles, had connected the disease with these glands, but did not make any microscopic investigations into its nature.

Before describing the alterations of the skin which I found in a case which I had the opportunity of observing, I will state shortly what was found by the authors whose names I have just mentioned.

Neumann,[1] who first published on this subject, describes the changes as consisting of alteration of the form and relative size of the papillæ, cell infiltration of the corium, and changes in the sebaceous glands. The changes in the glands consisted in growth of connective tissue around their walls, and degeneration of the secreting cells.

Geddings [2] examined skin which was taken from the back of a patient. In this woman knotty swellings in the subcutaneous tissue preceded the erythematous appearance on the skin, and this condition he considered to be the first stage of the disease. In the subcutaneous knotty swellings over which the skin seemed normal he found enlargement of the sebaceous glands caused by a swollen condition of the secreting cells. The vessels around the follicles were enlarged and distended with blood-corpuscles. The surrounding connective tissue seemed softened, and the spaces between the fibrillæ widened.

[1] 'Wien. Med. Wochenschrift,' 1863.

[2] 'Sitzungsb. d. k. k. Akad. d. Wiss.,' 1868.

In knotty swellings over which the skin was reddened, in addition to the appearances already noted, the capillaries in the papillæ were widened. When the diseased part was still nearer the surface and felt hard to the finger, in addition to the before-mentioned alterations of the sebaceous follicles and the surrounding connective tissue, the latter was thickly infiltrated with cells. At the stage in which crusts had formed the cell infiltration was so abundant that the various elements of the tissue could with difficulty be identified. In the stage of cicatrisation these cells were filled with fat-molecules and shrunk. He mentions also a slight cell-growth around the sweat-gland coil.

As the result of his observations he makes the inference that lupus erythematosus is a special form of inflammation of the skin which begins at the sebaceous follicles.

Kaposi[1] examined the affected skin in one of the cases in which the acute eruption had taken the form of hæmorrhagic vesicles. He found that the hair and sebaceous follicles were only slightly affected, more decided pathological changes being found in and around the sweat-glands.

The sweat-coil and duct were surrounded by abundant cell-infiltration; the cells of the duct were granular, and the duct itself blocked up and atrophied. Accordingly, he defines the disease as an inflammation that may take its departure either from the sebaceous follicles or the sweat-ducts.

In the spring of 1873 I had an opportunity of seeing a man who died at Vienna, in the wards of Professor Hebra, of the marasmus that sometimes supervenes in cases of lupus erythematosus of long standing, simultaneously with acute eruptions on parts of the skin previously healthy. He had suffered from the disease for eight years, chiefly on the face and scalp. The day after his death I obtained a small piece of skin for the purpose of examination, and I have Professor Hebra's kind permission to publish the results.

For a few weeks before the patient died, the eruption

[1] 'Archiv f. Derm. u. Syphilis,' 1872.

had appeared in a very decided form on the dorsal surfaces of the first phalanx of the fingers and toes. On the toes it was beginning to spread from the dorsum to the interdigital surfaces, and in order to be able to examine a diseased part in the earliest stage I cut out a piece of skin from the first toe at the part where the dorsum passes into the interdigital portion of the integument. After hardening for about a week in a solution of chromic acid, thin vertical sections were cut and examined. What I found was as follows:

The capillaries of the papillæ were enormously distended and filled with red blood-corpuscles. In some papillæ this distension was so great that a capillary loop nearly filled a papilla of medium width. In different parts of the corium, similarly distended and filled capillaries were also seen. The veins were also distended and filled with blood. The arteries were empty. The abundant network of capillaries surrounding the sweat-glands were also greatly distended and filled with blood-corpuscles.

The rete Malpighii, fibrillary tissue of the corium, and sweat-glands were perfectly healthy. I had cut so far beyond the region of the sebaceous glands, which, as is known, are here limited to the dorsal surface, that although I examined all the sections made from the portion of skin, in only one of them was there a hair and sebaceous follicle, and the gland was normal in appearance.

In regard to the sweat-glands of this part of the body I may mention that they are of a very large size, proportionate to the abundant secretion of sweat that takes place between the toes, and in some of the sections made from this man's toe the single layer of smooth muscular fibres that surround the larger sweat-coils were distinctly seen, and were also normal in appearance and development.

The importance of these facts when compared with those related by Neumann, Geddings, and Kaposi, and with the inferences they have made from them, is chiefly of a negative kind.

The great width and unbroken lumen of the capil-

laries of the papillary layer indicate a congestion of some standing, and yet the fibrillary tissue and secreting structures of the skin were unaltered in appearance. Whatever possible cell-infiltration existed was not such as was indicated by staining with carmine. The disease was thus firmly established in a part of the skin where there are no sebaceous glands, and in which the sweat-glands still presented a normal appearance.

But although none of the changes that have been described by the authors above named as occurring in the sebaceous and sweat-glands and fibrillary tissue were present, there was a morbid condition of the capillary blood-vessels which, if it had been persistent, was certain, in course of time, to have produced all of them. This morbid condition was naturally most apparent where the supply of capillaries in the skin is greatest, namely, in the papillæ and around the sweat-glands.

An atrophied condition of the papillary layer of the cutis —what Cazenave has described as a thinning of the skin— must be the inevitable consequence of a prolonged stasis by which the circulation is virtually destroyed.

The conclusion to which these results naturally lead is, that as yet we know in this disease no changes antecedent to the congestion of the capillaries, and can associate it with no special constituent organs of the skin. This conception of its nature seems to me to be especially consistent with the history of cases of acute eruption, and more especially with those in which the eruption consists of hæmorrhagic vesicles. I would suggest, therefore, that the affections of the sebaceous and sweat-glands should be considered, not as being necessarily the causes of the congestion, but as following it. A fuller investigation into the pathological anatomy of the disease is, however, urgently needed.

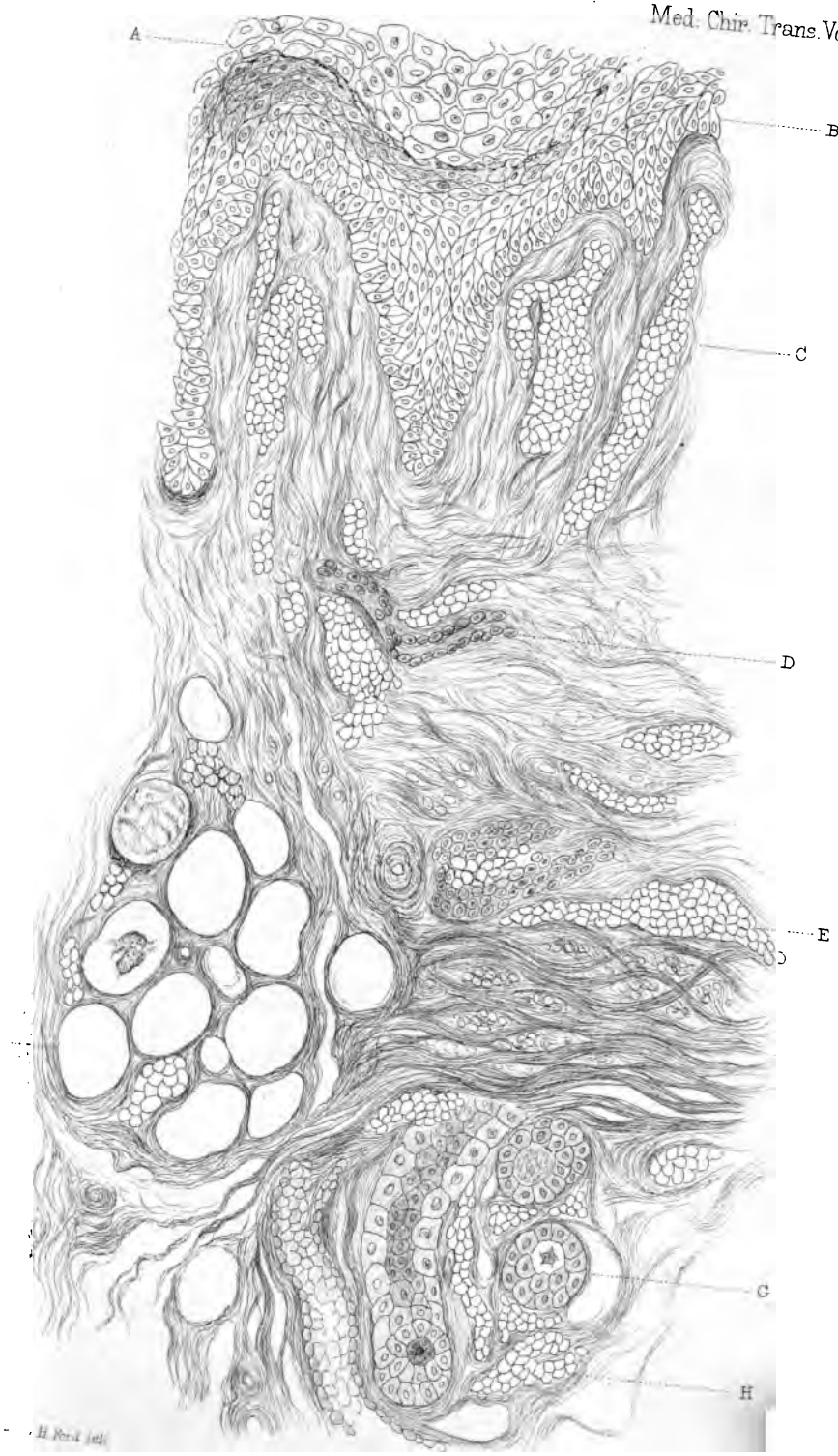
Without entering on the question of remedies I may point out, in conclusion, merely, that as the primary stage is coincident with distension of the capillaries, the promising treatment introduced by Dr. Veiel, consisting in puncturing the whole of the erythematous surface with an instrument com-

posed of a number of closely apposed lancets, is at least a rational one.

The accompanying drawing was made by Mr. Ewart from one of the sections preserved in Dammar varnish. The fat vesicles at the lower edge have been slightly displaced in order to limit the size of the drawing.

DESCRIPTION OF PLATE I.

A. Lower part of the horny layer of the epidermis.
B. Rete Malpighi.
C. Distended capillary of a papilla (blood-corpuscles).
D. Artery.
E. Distended capillary in the corium (blood-corpuscles).
F. Fat.
G. Section of sweat coil.
H. Distended capillary in sweat coil (blood-corpuscles).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ON THE TEMPERATURE

OF

PHTHISIS PULMONALIS,

AND ON THE

VARIOUS CONDITIONS INFLUENCING IT.

BY

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(Received December 10th, 1874—Read January 26th, 1875.)

The value of the thermometer in the clinical study of phthisis, whether as a means of diagnosis from other diseases, or between forms of the same malady, or, again, as a measure of the degree of fever and constitutional infection, is a subject which has been largely discussed both here and on the Continent, and the very various and often discordant views held by different observers lead one to believe that the true temperature-course of phthisis is yet an unsolved problem.

Jochmann[1] appears in 1853 to have arrived at a more elaborate classification of the temperatures in consumption than any who followed him. He enumerates three types, and to each of these he appends two or more variations; in the case of one type even these variations are subdivided again, and the impression derived from a perusal of this

[1] 'Beobachtungen über die Körper-Wärme in chronischen fieberhaften Krankheiten.'

careful arrangement is that the exceptions considerably outnumber the rules, and but little comfort is conveyed by Jochmann's further statement that all these types may alternate with another in a single case, or by Dr. Wilson Fox's objection that acute tuberculosis is not included in Jochmann's tables.

Dr. Sydney Ringer, in the first edition of his 'Temperature in Phthisis,' lays down some striking propositions deduced from twenty-four cases; among these we find "that a daily elevation of temperature probably takes place in all cases in which a deposition of tubercle is taking place in any organ;" also "that the temperature may be taken as a measure of the amount of tuberculosis and tuberculization, and fluctuations in the former indicate corresponding fluctuations in the severity of the disease." Dr. Ringer insists, too, that in many instances the temperature is of more value than physical signs and symptoms for the diagnosis of phthisis.

In his second edition these views are maintained with some modification, and whilst admitting that tuberculization or catarrhal pneumonia may advance so slowly as to cause little or no elevation of temperature, he states that where this symptom is absent we may conclude that the progress of the disease is almost insignificant.

All workers on the subject must feel greatly indebted to Dr. Ringer for his additions to our knowledge, and for the many valuable suggestions which he has made for future inquiry; and if the cases I am about to bring forward have led me to differ from him in some respects, in many others they have, from larger data than his own, abundantly confirmed his conclusions.

Niemeyer[1] held that the thermometer enabled us to distinguish between the tuberculous and pneumonic forms of phthisis, because of the greater remissions displayed in the charts of the former, while on the other hand several observers, including Wunderlich and Lebert, maintain that no thermometrical distinction is possible between the anatomical forms of the disease.

[1] 'Klinische Vorträge.'

Lebert,[1] in a laborious treatise based on observations on several patients, extending over many years, gives a number of careful results, and states his conclusions in the small number of fifty-one propositions! many of which, when carefully compared, are found to contradict one another, and his most important one, viz. "that individual idiosyncrasy influences the temperature course more than the form, localisations, or phase of the disease," does not add to the lucidity of his deductions or afford a very safe ground for classification.

Dr. Wilson Fox has made the most recent contribution to the subject in the form of a lengthy and painstaking paper in the fifty-sixth volume of the 'Medico-Chirurgical Transactions.' His observations were made twice a day, morning and evening, on eighty in-patients at University College Hospital, the cases being "instances of more or less distinctly advanced disease admitted to a general hospital on account of its acuteness, its extent, or recent aggravation." Dr. Fox acknowledges that they represent severe cases only, and "thus afford no information respecting the earlier stages, or the periods of comparative quiescence."

Dr. Fox's *cases* are divided into—1st, *fatal*, including acute tuberculosis, acute pneumonic phthisis, and chronic phthisis; 2nd, *non-fatal*, subdivided into high and low temperatures.

Both these classes seem to me based on somewhat arbitrary principles, as the class in which a patient would be placed depended on the part of his career which he spent in the hospital; also the division into high and low temperatures seems to involve a *petitio principii*, as it ought to be the author's object to arrive at a clinical or pathological basis of thermal classification, and not to prejudge the matter by dividing into classes *according to* the temperature. After a minute analysis of the temperatures in all these classes—in which, by-the-bye, little or no note is taken of the physical signs, and the extent of consolidation or excavation—he comes to no very definite conclusions beyond agreeing with Lebert, "that difference of temperature depends either on the con-

[1] 'Deutsch. Archiv Klin. Med.,' Nov., 1872.

stitutional peculiarity of individual patients, or on the varieties in the pyrogenic effects of the changes in the lungs which require further elucidation."

A careful survey of the above authorities impressed me with the conviction that the observations hitherto made have been carried out in the routine fashion generally in vogue in hospitals for exanthematous fevers, surgical inflammations, and the like, and that sufficient notice has not been taken of the peculiarities of the special disease under consideration.

By the majority of observers the temperature was taken only twice a day, morning and night, even the hours not being always specified, and the changes in the intermediate period remaining an untold tale, though one authority speaks of remissions from morning to evening, and the reverse, as if the thermometer stood still and the course of events between these periods was unchequered by variations.

It is obvious that many and great changes may take place between these two periods, and observations may show a case to be pyrexial in the afternoon, whose morning and night records are perfectly normal, and, indeed, Kuchenmeister[1] maintains that the maximum exacerbation occurs between noon and 2 p.m., and that a lowering follows till 5 p.m., when a second exacerbation occurs much inferior in intensity to the midday one. No note, moreover, is found in any of the above authorities of the thermometric changes occurring during the hours of rest, or the influence of sleep on them, and it would naturally occur that this would be the proper period to mark the commencement of pyrexia, or the subsidence of the same.[2]

A second great objection presents itself in the fact that the observations have been made on the disease as a whole, and not on its individual stages and developments. Surely some regard should be had to physical signs, and obser-

[1] 'Kaltes Wasser,' p. 220.

[2] Dr. Wilson Fox admits that on this account his results have only an approximate value, and I understand that both he and Dr. Ringer are now modifying their plan, and taking observations oftener. Some of Dr. Ringer's original cases were taken three or four times daily.

vations taken when these indicated the formation of tubercle should not be jumbled up with others recording the progress of a discharging cavity. Or, again, the records of intercurrent inflammations should not be entirely confused with others of progressive excavation. In the above instances the formation of pus should be duly taken into consideration, as its absence or presence may entirely modify the temperature course.

The unusual opportunities afforded by my position as physician to the Hospital for Consumption and Diseases of the Chest, Brompton, where I have from fifty to sixty beds under my charge, enabled me to institute a more complete series of observations than has ever been before attempted; for within these wards may be found at the same time instances of all stages and degrees of consumptive disease subjected to the same hygienic conditions.

One great advantage this institution offers for thermometric investigations is that by means of the heating and ventilating apparatus an equable temperature of from 60° to 63° Fahr. is maintained day and night, and in order to exclude the effects of recent colds, fatigue, inanition, and other disturbing elements, it has been my practice not to commence observations until the patients have been at least a week in the hospital, and therefore placed under circumstances more likely to ensure accurate results.

The number of patients furnishing the results was 111; 66 males, 45 females; nearly all adults. The ages being from 13 to 49; the average of the males being 26, and of the females 27.

The greater number of the observations were made in the axilla, the thermometers being left in about ten minutes. Mouth temperatures were taken in a few cases, and in one a double record of both mouth and axilla heat was made with a view of checking the general results and of testing the accuracy of other observers on this point.

The thermometers used were principally those of Mr. Hawkesley, and were found, on testing, to be very accurate; a few furnished by other well-known makers, as Mr. Casella,

Messrs. Harvey and Reynolds, of Leeds, and Messrs. Meyer and Melzer, were also in use.

The observers were, for the most part, the various gentlemen who have held the offices of assistant resident medical officer or of clinical assistant at the Brompton Hospital during the last four years, viz. Messrs. Bartlett, Parry, McKinlay, Williams, Hartley, Peacey, Crocker, Boys, Murrell, Bernays, and Kelly, superintended and directed by myself; and I take this opportunity of gratefully acknowledging the careful and steady manner in which the observations were made and my directions carried out, involving, as may be seen hereafter, considerable sacrifices of time and comfort. It was only by infusing into my companions and myself a spirit of fervid scientific enthusiasm to attempt the solution of the oft-vexed problem of the temperature in phthisis that we succeeded in enduring the irksomeness entailed by so long and tedious a series of observations.

The patients were, for the most part, under my own care, but as my colleagues kindly allowed me to make use of any of their cases of interest bearing on the subject, I did not hesitate to avail myself of special opportunities occurring in their wards.

The hours of observation at first selected were 8 a.m., 11 a.m., 2 p.m., 5 p.m., and 8 p.m., and in none of the 111 cases were the temperatures less often taken than at these five periods.

The number of days during which this record was carried on varied from seven to thirty, excepting three cases of low temperature, where days registered did not reach seven.

In the course of these observations so much irregularity was noted that I deemed it necessary to investigate the temperatures of intermediate hours, and, therefore, supplementary observations were taken at 10 a.m., at 3, 6, 7, 9, 10, and 12 p.m. in many of the cases, and in this way 4226 records were collected. Thus we arrived at some definite conclusions with respect to the day temperature of phthisis; but to test these conclusions especially with regard to cavity cases, hourly observations were taken in one instance, in a

well-marked third-stage patient, from 8 a.m. to 8 p.m., thirteen times a day for a period of a fortnight, by Mr. McKinlay (see Plate II).

Our next step was to investigate the temperatures of the night and of the early morning, and to compare these with the day records, and for this purpose I selected twelve of the above-mentioned patients in whom the different stages of the disease were well marked, and in all of these, except one, an hourly record was carried out for twenty-four hours; in that one the twelve night hours only were noted.

In two of these cases, in order to confirm the first thermic cycle, a second series of hourly temperatures for a day and night were recorded.

At the same time careful notes were taken of all phenomena which could possibly influence or be influenced by the temperature, *e. g.* the state of the pulse, respiration, skin, bowels, the quantity of urine passed, the hours of meals and the food taken, the administration of drugs, the amount of sleep, and the temperature of the ward.

This laborious inquiry was carried out by my kind assistants, Messrs. McKinlay, Williams, Parry, Boys, Murrell, and by myself. A specimen of our mode of record is exhibited.

Charles R—, æt. 17.

Date.	Hour.	Temp.	Pulse.	Resp.	Meals.	Temp. of ward.	Notes.	Physical signs.
June 17	8 a.m.	98·8°	104	32	...	64°	Awake; skin moist; urine plentiful and normal	*Right.*—Flattening; crepitation, whole front, most marked and coarse at base. *Left.*—Very coarse crepitation, lower half; cavernous sounds between fifth and sixth ribs.
	9 a.m.	100·2°	120	36	Breakf'st, egg, bread, butter, and cocoa	„	...	
	10 a.m.	103·2°	120	32	...	„	Skin burning hot; complains of thirst	

An account of the principles which have guided my arrangement of cases must now be given, and I need hardly say that my object has been to simplify these as much as possible.

Any strict classification of cases of consumption is attended with great difficulties, and cannot be carried out without making arbitrary distinctions which neither clinical nor pathological observations support. Whilst fully recognising the different parts played by the inflammatory and infective processes in phthisis, I have always maintained that the various forms are mere groupings of the same terrible disease, and that between these groups every gradation of link, pathological and clinical, can be found.

Now that we have cleared our eyes from the mists of Niemeyerian doctrines, and contemplate phthisis as it exists ordinarily and not exceptionally, the unity of the disease appears as well founded as it did before the cloud of German speculation obscured the horizon.

The most valuable result of the debate on Tubercle at the Pathological Society has been to establish a distinct anatomical basis for most forms of phthisis, and to demonstrate the identity of the grey granulation, the soft granulation, and the various caseous changes by the presence of the small-celled lymphoid growth; and if we owed Dr. Wilson Fox no other debt of gratitude, this alone of having settled the tubercle question on a basis so satisfactory to future workers would entitle him to our warmest thanks.[1]

The cases on which the temperature observations were made were of the ordinary type of consumptive patients as admitted into the Brompton Hospital, and except that some may be called acute and others chronic cases, it would be difficult to separate them abruptly.

They were no instances of rare forms of tuberculosis requiring microscopical investigation for its detection, but

[1] At the same time we must not forget the considerable part played by *exudation* in many acute cases of phthisis. Dr. C. J. B. Williams has long held this to be the case, and recently Dr. T. H. Green has stated that the small-celled growth is absent, and exudation and epithelial proliferation are the chief characteristics of many cases of acute phthisis (not tuberculosis).

rather examples of that common and deadly disease familiar to us all, and fatal yearly to one eighth of our population.

One hundred and four of the patients under observation have been arranged in five classes, according to the stage of the disease and the activity of its progress: 1st stage, *active*, 26 cases; 1st stage, *quiescent*, 5 cases. 2nd stage, 10 cases. 3rd stage, *active*, 43 cases; 3rd stage, *quiescent*, 20 cases.

This arrangement is, of course, almost entirely based on physical signs, and in the 10 cases in which post-mortem examinations were made the diagnosis was amply confirmed.

As will be seen, the relations of *temperature* to *the formation of tubercle*, to *softening* and *excavation*, and again to continued ulceration of the lungs, are all carefully worked out.

It is because physical signs are our chief means of detecting the occurrence of the above phenomena that I have made them the basis of my divisions, but as far as possible full regard has been paid to clinical and pathological features, always excepting the temperature itself, as it was my object to classify, not it, but its accompanying conditions, and thus to deduce its factors.

In the 104 patients both lungs were attacked in 66 (almost two thirds), one lung in 38 (about one third), the right alone in 17, and the left alone in 21. Of the 31 in the first stage 16 had both lungs affected, and 8 a single one; of the 10 second-stage cases 5 had both lungs involved, and 5 one lung only; and of the 63 in the third stage 45 had both lungs attacked, of which 10 had double cavities.

We will now proceed to consider each class of cases and the temperature prevalent in them, and first let us take Class I, *first stage, active* (Plate III, fig. 1).

The physical signs generally noticed here were (1) those indicating the commencement of disease in the lung hitherto free, or (2) the extension of disease in a lung part of which was already consolidated; and great care has been taken to exclude all cases where anything besides consolidation or scattered tubercle was suspected. The crepitation was generally of a fine description, and, as regards locality, often

scattered over the upper lobes. That it did not pass into the coarse crepitation of a croaking character typical of softening was confirmed by subsequent observation in each instance.

The following instance of the *development of disease* and its accompanying thermal phenomena will help to illustrate the features of this class:

Case 1.—W. G. W—, æt. 14, was admitted into Wallace Ward, April 29th, 1873.

History. — Father died of consumption. Sister suffers from chest disease.

A week after Christmas, was attacked with giddiness and pain in right chest, and night sweats; three days after he complained of cough with scanty expectoration, which has continued since; has not lost much flesh; night sweats have persisted since Christmas; complains of cough and dragging sensations in the right shoulder; bowels costive; tongue furred; appetite fair; skin rather anæmic; pulse 136; temp., 8 p.m., 102° F.; weight, May 8th, 6 st. 12½ lbs. He was carefully examined on May 5th, and no physical signs could be detected. The temperature was taken five times daily, and showed the usual pyrexia of first-stage phthisis, *i. e.* lower or normal records at 8 a.m., slightly raised at 11 a.m., and high after 2 p.m.

On May 8th dulness was detected over the left clavicle and crepitation below it.

	8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.	
Saturday, May 5th .	98·8	100·	100·6	101·	100·2	No physical signs
May 6th . . .	98·4	98·2	100·6	101·	101·4	
May 7th . . .	98·3	99·	100·4	100·2	100·	
May 8th . . .	99·4	99·8	101·	102·	101·3	Dulness detected over the left clavicle, and crepitation below it.
May 9th . . .	98·	99·4	100·	100·5	101·3	
May 10th. . .	98·2	99·	99·6	101·	100·4	
Average . .	98·5	99·2	100·3	100·9	100·8	

June 26th.—Dulness of the whole left side; crepitation to third rib; crepitation above right scapula.

July 17th.—Crepitation to fourth rib on left side; coarse under clavicle; weight, 7 st. 1 lb., having never fallen since admission.

This case was a well-marked instance of the development of tubercle, and, as the rise in temperature preceded the appearance of physical signs, was confirmatory of Dr. Ringer's observation that we can often diagnose tubercle by the temperature before we can do so by physical signs.

The effect of *extension of disease* in the same lung is exemplified by Case 2, where the crepitation which had been confined to the apex of the lung extended to the whole of the lung (while the temperatures were being taken), and at a later date to both lungs. It will be observed that though there is a rise in the afternoon, yet with all the active disease going on the temperature does not rise high, and only twice reaches 100° F.

Case 2.—Richard D—, æt. 25, admitted into the Brompton Hospital, December 23rd, 1872.

History.—Mother and brother died of consumption; cough with expectoration two and a half years, succeeded six months later by an attack of slight pleurisy, followed by night sweats and wasting. Night sweats disappeared, but have returned within the last six months and are now profuse. Has wasted more or less for six months. Hæmoptysis to the amount of ℥j eighteen months ago, and the expectoration has been streaked on several occasions.

At present.—Cough troublesome; tongue clean; appetite fair; bowels regular; pulse 80; resp. 24.

At the end of the first week of January the physical signs were "crepitation to fourth rib on the right side." The subjoined temperatures were then taken.

January, 1873.	8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.
8th	98·	98·2	99·2	99·3	100·2
9th	97·	98·	98·8	98·4	99·6
10th	97·2	98·8	99·5	99·4	99·3
11th	97·1	98·6	99·4	99·6	99·4
12th	97·	97·8	98·6	99·2	99·4
13th	96·8	98·1	99·1	99·4	99·6
14th	96·8	98·2	99·	99·4	100·2
Average	97·1	98·2	99·1	99·2	99·5

January 30th, 1873.—Crepitation extends over the whole right front, and over the right interscapular region.

March 6th.—Crepitation has diminished in the upper portion of the right lung, but is now audible, scattered throughout both lungs front and back.

The above cases demonstrate the effect of tubercle formation in causing a definite rise in the temperature, which may cease when this process is complete. It is, however, by no means necessary that any rise should take place, and in several of my cases, where active disease has been going on in one or both lungs, no rise has taken place, but rather a low range of temperature has prevailed. Of this the subjoined is an instance.

Case 3.—Janet S—, æt. 46, admitted into the Brompton Hospital, December 2nd, 1873.

History.—Subject for the last four years to cough in winter, which for the last two years has been continuous, and accompanied by loss of flesh. Night sweats for one year; slight hæmoptysis four years ago, and again, two years ago she brought up a pint of blood.

At present.—Cough troublesome, sputum yellow and frothy; night sweats profuse; pains under both clavicles, most under right; tongue clean; appetite good; bowels regular.

On the first of January the physical signs were—

Right.—Dulness; crepitation upper third, front, and back.

Left.—Crepitation audible over whole front and over interscapular region.

The following temperatures were taken two days later:

January.	8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.
14th . . .	98·	98·2	98·8	98·6	99·
15th . . .	97·	98·	98·3	98·	98·4
16th . . .	98·2	98·	97·8	98·	98·2
17th . . .	97·6	98·	98·6	98·6	99·
18th . . .	97·9	97·	98·4	98·	98·8
19th . . .	98·	98·2	97·6	98·4	98·4
20th . . .	97·6	98·	98·6	98·4	98·2
Average .	97·7	97·9	98·	98·3	98·6

January 5th.—Hæmoptysis to the amount of ʒiss; no increase of cough followed.

19th.—Profuse night sweats.

30th.—Physical signs the same.

Upwards of 200 observations on the class of *first stage active* were made in twenty-six patients, at the hours 8, 10, 11 a.m. 2, 5, 8, and 11 p.m., and the results are embodied in Table I, which records, under the various temperatures, the number of times each was noted, as well as the relative percentages. The last five columns express the *maximum, mean,* and *minimum* temperatures, taken at each hour, as well as the *number of cases,* and number of observations made on them.

Attention is especially drawn to the fifth and twelfth columns, as these contain the totals of observations of the pyrexial (100° and over) and of subnormal (under 98°) types, with their respective percentages, and a glance at these will give the reader of this and the succeeding tables at once the gist of the contents.

TABLE I.—*Analysis of Temperatures.*

First Stage (Active).

Hours.	103°.	102°.	101°.	100°.	100° and over	99°.	98°.	97°.	96°.	95°.	94°.	Under 98°.	Maximum.	Mean.	Minimum.	No. of observations.	No. of cases.
8 a.m.	...	...	...	2	2	10	76	70	19	2	1	92	100·	97·8	94·2	180	26
Percentages... ...	...	...	...	...	1·1	5·56	42·23	...	...	...	...	51·12	...	...	...	...	...
10 a.m.	...	...	1	6	7	2	3	8	1	...	...	9	101·5	98·6	96·	21	3
Percentages																	
11 a.m.	...	...	1	9	10	43	82	21	4	1	...	26	101·1	98·5	95·	161	23
Percentages... ...	...	...	...	...	6·21	26·70	50·93	...	...	...	...	16·14	...	...	...	...	...
2 p.m.	1	3	12	41	57	56	55	15	2	...	...	17	103·	99·3	96·	185	
Percentages... ...	...	...	...	22·16	30·81	30·27	29·72	...	...	...	...	9·18	...	...	...	...	26
5 p.m.	2	8	15	44	69	52	53	9	2	...	...	11	103·4	99·4	96·	185	...
Percentages... ...	...	...	...	23·78	35·67	28·11	28·64	...	...	...	...	5·94	...	...	...	...	26
8 p.m.	...	9	18	56	83	58	44	5	...	...	...	5	102·5	99·6	97·5	190	...
Percentages... ...	...	...	...	29·47	43·68	30·52	22·10	...	...	...	...	2·63	...	...	...	...	26
11 p.m.	...	...	1	1	2	6	7	1	3	...	2	6	101·	98·3	94·	21	...
Percentages... ...	...	...	...	...	9·52	28·57	33·34	...	...	...	...	28·57	...	...	...	...	3
																	...

By this method I hope to put the Fellows of the Society in full possession, not only of the results, but also of the facts on which these results are based. The observations taken at 8 a.m. exhibit remarkably low temperatures; 51 per cent. are below 98°, 42 per cent. between 98° and 99°, while only 5½ per cent. rise to between 99° and 100°, and 1 per cent. reaches 100°; and as this last temperature (100°) was only recorded in one case on two occasions, it may fairly be considered as accidental.

The mean temperature is 97·8°, while the minimum falls as low as 94·2°.

The notes at 10 a.m. show a slight rise from the subnormal state, which is more marked at 11 o'clock, when we find only 16 per cent. below 98°, 50 per cent. between 98° and 99°, and 6 per cent. above 100°; the mean temperature being 98·5°, the minimum 95°. The 2 p.m. and 5 p.m. observations show a steady continuance of this rise, the subnormal temperatures diminishing and the pyrexial increasing, until at 8 p.m. the climax is reached, only 2½ per cent. being subnormal, and more than 43 per cent. exceeding 100°; the maximum being 102·5°, the mean 99·6°, and the minimum 97·5°.

The 11 p.m. notes show a rapid fall, 28½ per cent. being subnormal, 33 per cent. normal, and only 9½ per cent. above 100°. The dotted line (see Plate III, fig. 1, No. 1) expresses roughly the day-temperature course of Class I as far as can be ascertained from observations taken seven times per diem in these twenty-six cases; but in order to display the night phenomena and to give a fuller insight into the day variations a second continuous line (see Plate III, fig. 1, No. 2) has been annexed which shows twenty-four hourly observations on one of the same patients, and this may be compared with the dotted one.

Though this case was hardly a typical one, the general course may be said to confirm the dotted line, especially in respect of the rise, which, it would appear, dates from the morning hours. It shows a slight exacerbation at 10 a.m. and a consequent fall, and then a nearly continuous though

slight rise from 2 p.m. till 10 o'clock, when a rapid fall commences, which reaches its lowest ebb between 2 and 3 a.m. Temperatures of 94° and 95° are then not unfrequently recorded.

The next class, Class II, is that of *the first stage, quiescent* (Plate III, fig. 2).

This is the ordinary "chronic first stage" of authors, of which an instance is subjoined, though its common occurrence in practice renders it familiar to every one.

Case 4.—Julia E—, æt. 21, admitted into Hall Ward under Dr. Quain, April 14th, 1874. Maternal aunt died of phthisis. Cough with expectoration and wasting six months; worse for four months; hæmoptysis, ʒj, four months ago, and to slighter amount once since. Night sweats occurred at the same period, but have since ceased. Appetite bad; tongue furred; catamenia scanty and irregular for nine months; bowels confined; pulse quiet.

Physical signs, May 13th, 1874.—Dulness and tubular sounds over upper third of right lung; no night sweats; expectoration slight.

The subjoined observations were then taken:

Date.	8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.	11 p.m.	Pulse.
May 14th . . .	...	98·2	98·4	...	98·	...	64
„ 15th . . .	97·6	98·2	98·4	97·	97·4	...	64
„ 16th . . .	98·4	97·6	97·2	97·2	98·6	...	64
„ 17th . . .	97·4	97·	96·2	97·6	97·4	97·2	60
„ 18th . . .	98·4	97·4	97·8	98·2	97·6	...	72
„ 19th . . .	97·4	97·8	98·2	97·	97·4	...	60
„ 20th . . .	97·2	97·2	97·	98·6	97·	...	56
„ 21st . . .	97·4	97·	99·6	101·2	99·4	...	68
Average . . .	97·7	97·5	97·8	99·5	97·8	...	...

May 22nd.—Physical signs the same.

No marked change is indicated by the 8 o'clock records, when the mean is 98·4°; after this period a distinct fall

takes place, which, as in the acute first stage, continues till the early morning hours. I have made a series of hourly observations in one case for twenty-four hours to gain some insight into the night temperatures, and the result is seen in the continuous line.

The patient was a male, æt. 36, with well-marked physical signs of consolidation over the upper third of the left lung, and no advance in the signs had been detected during the two months he had been under observation. The temperatures are chiefly normal in character, with the slight tendency to afternoon rise which seems to be often present. The continuous line in this diagram (Plate III, fig. 2, No. 2) gives the cycle in this instance.

Table II is based on between thirty and forty observations made on five patients of this class at each of the following hours, viz.—8 and 11 a.m.; 2, 5, and 8 p.m.; a few were made at 11 p.m. which confirm the general course (see page 84).

The 8 a.m. observations indicate subnormal temperatures, the mean being 97·3°, the minimum 95·2°, and the maximum 98·5°; 63 per cent. fell below 98°, and the remainder are all below 99°. At 11 o'clock the temperatures are still low, the average being 97·9°, the minimum 95·2°. There is a slight increase, however, in the percentages under the heads of 98° and 99°.

The further notes show a steady rise at 2 p.m., which reaches its maximum at 5, an average of 98·7° being then attained, for though at 2 p.m. 8⅓ per cent. exceeded 100°, the average was only 98·6°.

The next class is *second stage* (Plate III, fig. 2, No. 3), and under it are included only cases where no cavity has hitherto existed, but where the course of the physical signs clearly demonstrates one to be in process of formation. The signs principally relied on have been the increasing coarseness of the crepitation, the hoarse and croaking rhonchus, confined to a portion of one lung and combined with tubular sounds, and often high-pitched percussion note. In most cases the large increase of expectoration and the detection of elastic tissue

Table II.

First Stage (Quiescent).

Hours.	103°.	102°.	101°.	100°.	100° and over.	99°.	98°.	97°.	96°.	95°.	94°.	Under 98°.	Maximum.	Mean.	Minimum.	No. of cases.	No. of observations.
8 a.m.	...	...	...	...	...	...	12	13	5	3	...	21	98·5	97·3	95·2	5	33
Percentages	...	...	...	...	...	...	36·36	...	...	...	...	63·63	...	...	...	...	...
11 a.m.	...	...	...	...	...	6	16	8	2	3	...	13	99·6	97·9	95·2	5	35
Percentages	...	...	...	...	...	17·14	45·71	...	...	...	...	37·14	...	...	...	...	...
2 p.m.	...	...	...	3	3	11	12	8	2	...	...	10	100·4	98·6	96·2	5	36
Percentages	...	...	...	...	8·33	30·55	33·33	...	...	...	...	27·77	...	...	...	...	...
5 p.m.	...	...	1	1	2	11	10	9	2	...	..	11	101·2	98·7	96·4	5	34
Percentages	...	...	...	...	5·88	33·35	29·41	...	...	...	...	32·35	...	...	...	...	...
8 p.m.	...	...	...	2	2	13	18	14	...	...	...	14	100·4	98·4	97·	5	37
Percentages	...	...	...	...	5·40	35·16	21·62	...	...	...	...	38·	...	...	...	...	...

in it have confirmed the physical signs. In many of the cavity cases, which we shall presently consider, softening was proceeding in the opposite lung, or in another part of the same lung, but here, as a suppurating surface already existed in the form of a cavity, which might considerably influence the temperature, it was deemed advisable to separate these cases from other cases of softening and to class them under the heading "Active third stage."

We will now consider the process of softening in its thermal relations.

The observations were made on ten patients, at the same hours as in the preceding class, between seventy and eighty records being taken at each period, and of these, two examples, where the physical signs can be compared with the temperatures, are annexed. In the first one (Case 5) the formation of a cavity was proved by physical signs and the presence of yellow elastic tissue in the expectoration, the excavation being accompanied by a rise of temperature. In the second (Case 6) excavation was proved by physical signs alone, and gave rise to no pyrexia.

Case 5.—Elizabeth B—, æt. 28, single, admitted into the Hospital for Consumption, January 21st, 1873. Mother died of phthisis. Subject to winter cough for six years, worse last winter, and continuous since. Dyspnœa for seven months. Slight hæmoptysis and night sweats last May, and has been losing flesh ever since. Catamenia ceased in May, but have reappeared in October, and are now regular. Cough troublesome, with expectoration; pulse 80. Crepitation is audible over the upper third of the left front chest.

February 17th, 1873.—Cough worse, with muco-purulent expectoration; respirations 32; croaking crepitation audible in the first interspace on the left side; coarse crepitation from the second to the fourth rib; croaking crepitation in the upper half of the left back. The subjoined temperatures were then taken.

Date.	8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.
February 18th . . .	98·	99·	99·2	100·	101·4
„ 19th . . .	98·6	99·	99·	100·	101·
„ 20th . . .	99·6	99·6	99·	100·2	100·
„ 21st . . .	98·2	99·	98·6	99·8	100·8
„ 22nd . . .	97·8	100·4	99·4	98·6	98·8
„ 23rd . . .	98·	99·2	99·	100·2	100·2
„ 24th . . .	98·	98·	100·6	98·8	99·
Mean	98·3	99·2	99·3	99·7	100·2

20th.—Yellow elastic tissue detected in the sputum, which is abundant, amounting to half a pint daily.

March 5th.—Well-marked cavernous gurgle in upper left chest.

April 15th.—Dry cavernous sound to the second rib; some croaking respiration above the scapula; cough easier; expectoration small in amount. Has lost 6½ lbs. during her stay in the hospital.

Case 6.—Edwin N—, æt. 23, carpenter, admitted into the Hospital for Consumption, January 29, 1873.

History.—Continuous cough for one year. Slight hæmoptysis last May, accompanied by pain in the left chest, and has been wasting ever since. Night sweats on and off since May, with dyspnœa on exertion. Tongue clean; appetite bad; bowels regular; pulse 100; respirations 24; cough troublesome.

On admission the physical signs were dulness and crepitation in the first interspace on the left side.

On March 13th the disease had extended, and crepitation was audible over the upper half of the left chest posteriorly. Crepitation was discoverable over the whole left front, and was of a croaking character in the upper half.

The temperature was then taken five times a day, and the subjoined is the chart.

Date.	8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.
March 20th . . .	98·8	99·4	99·6	98·4	97·6
„ 21st . . .	98·	99·	97·	99·	98·
„ 22nd . . .	98·	99·	98·8	98·6	98·2
„ 23rd . . .	95·6	98·	98·	98·2	98·
„ 24th . . .	96·	98·6	99·	99·4	100·
„ 25th . . .	97·	99·	98·2	99·	99·
„ 26th . . .	98·	98·2	98·4	98·2	99·2
„ 27th . . .	97·4	98·	98·6	...	...
Mean	97·4	98·6	98·4	98·7	98·5

March 27th.—Cavernous gurgle detected in upper left front. Patches of urticaria have appeared on the patient's body.

Let us now consider the statistics of the second stage as set forth in Table III (see page 88).

At 8 a.m. the temperatures are somewhat higher than in the first stage, 37½ per cent. being below 98°, 42½ per cent. between 98° and 99°, and 5 per cent. exceeding 100°; the mean being 98·1°, and the minimum 95·6°.

At 11 o'clock a rise has taken place, the temperatures exceeding 100° being 27½ per cent., and those below 98° 10½ per cent.; the mean 99·1°, the minimum 96·8°.

A steady rise appears to take place till 8 p.m., the percentages above 100, at 2, 5, and 8 p.m., being respectively 40, 42¾, and 52, and the means 99·6°, 99·5°, and 99·7°; the highest temperature recorded, 103·6°, being noted at 8 o'clock.

The chart of this class has been depicted with open crossed dots (see Plate III, fig. 2, No. 3), and seems to indicate a continuous ascent during the day. Unfortunately I have not been able hitherto to observe many of the night temperatures, and therefore this cycle is at present incomplete.

The day observations, however, resemble those of the first stage in the low morning temperatures, but differ from them in exhibiting a more continuous rise from 8 a.m. to 8 p.m.

Table III.

Second Stage.

Hours.	103°.	102°.	101°.	100°.	100° and over.	99°.	98°.	97°.	96°.	95°.	94°.	Under 98°.	Maximum.	Mean.	Minimum.	No. of cases.	No. of observations.
8 a.m. . . .	...	...	1	3	4	9	35	23	5	1	...	29	101·4	98·1	95·6	10	77
Percentages . .	...	...	...	...	5·2	11·7	42·85	...	...	...	...	37·66	...	...	...	...	...
11 a.m. . . .	...	2	8	11	21	23	26	6	2	...	...	8	102·4	99·1	96·8	10	76
Percentages . .	...	...	...	...	27·63	30·26	34·21	...	...	...	...	10·52	...	...	...	...	...
2 p.m. . . .	...	4	15	12	31	26	15	4	1	...	...	5	102·5	99·6	96·4	10	77
Percentages . .	...	...	...	...	40·26	33·76	18·	...	...	...	...	6·5	...	...	...	...	...
5 p.m. . . .	...	1	10	16	27	13	17	6	...	...	...	6	102·	99·5	97·2	10	63
Percentages . .	...	...	...	...	42·85	20·63	27·	...	...	...	...	9·52	...	...	...	...	...
8 p.m. . . .	2	3	10	24	39	15	18	2	1	...	...	3	103·6	99·7	96·2	10	75
Percentages . .	...	...	...	...	52·	20·	24·	...	...	...	...	4·	...	...	...	...	...

The *third stage active class* (Plate IV, fig. 1) has been subjected to more thermometrical observations than any other, because the clinical and pathological phenomena which distinguish it are more distinctly phthisical than those of other stages. The crepitation accompanying tubercle formation may be mistaken for that of acute or chronic pneumonia, though the variations in the râles and their more scattered nature ought to give the key to a diagnosis. The rhonchus and sibilus which often usher in tuberculosis may be confused with asthma or bronchitis, until the serious character of the phenomena forces itself into notice, but the physical signs and clinical symptoms of an active third stage case are unmistakable. The large amount of purulent expectoration, the hectic aspect, the wasting, the night sweats, combined with the existence of cavernous sounds in the upper portions of the lungs, are referable to nothing else than a phthisical cavity discharging matter from its surface, or extending itself by further excavation, this being rendered indisputable by the presence of lung-tissue in the sputum.

The position of the signs, the character of the expectoration, and other clinical phenomena, at once separate cases of bronchial dilatation from this category.

While, however, it is easy to decide on the existence of a cavity, and to assign many of the symptoms to suppuration going on in the lung, a difficulty arises when in addition to this there is evidence of fresh tubercle forming either in the same or in the opposite lung. The question then arises, to which are we to assign the thermometrical phenomena—to the abscess symptoms, if I may so call them, or to the tubercle symptoms ?

It is hard to say, though probably the purulent processes exercise the greater influence, but a comparison between the *first stage active* and *third stage active* may throw some light on the subject.

I wish, however, to be clearly understood that in some of these forty-three cases the discharging cavity was accompanied by an increasing amount of tuberculization.

I subjoin an illustrative case of this class, in which

observations were taken seven times a day for twenty-two days, and shortly before the patient's death for two series of twenty-four consecutive hours.

Case 7.—Maurice A—, æt. 22, married, was admitted into the South branch of the hospital, April 7th, 1874.

History.—Had lost some maternal aunts from phthisis. Had suffered from general weakness for eight months, for four had had cough with expectoration, slight loss of flesh, and night sweats. Complains of pain in the back; indigestion after meals. Is very anæmic. Cough very troublesome at night, expectoration muco-purulent. Tongue furred; appetite moderate.

Physical signs.—Cavernous sounds audible to the fourth rib, right front. Weight (April 13th) 9 st. 7 lbs.

April 30th.—Has gradually become worse. Temperature and pulse have risen, and appetite has fallen off. Weight 9 st. 4 lbs. (a loss of 3 lbs.). In addition to above signs crepitation is audible under *left* clavicle, and tubular sounds in second intercostal space. Transferred to the main hospital in order to be more carefully attended to, as he is now confined to bed.

The following observations were then taken:

May.	8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.	Resp.	Pulse.	Physical signs.
8th.	98·4	100·	100·	100·	100·	30	104	May 7th.—*Right*: cavernous sounds to 4th rib, and over lower third. Crepitation in *left* upper chest.
9th.	99·9	99·8	98·8	98·5	100·2	...	...	
10th.	98·9	99·8	100·2	100·2	99·2	...	...	
11th.	99·4	99·8	100·2	100·6	100·6	32	100	
12th.	99·9	99·4	100·6	98·9	100·6	34	104	
13th.	97·8	100·2	99·9	99·4	100·4	36	112	
14th.	100·2	102·	100·2	100·	101·2	...	...	

The hours for taking the temperature were then changed to 11, 2, 5, 8, 10 and 12.

	11 a.m.	2 p.m.	5 p.m.	8 p.m.	10 p.m.	12 p.m.	Resp.	Pulse.	Physical signs.
May 23rd . .	99·9	100·2	100·1	100·8	101·3	...	...	...	In addition to the above, crepitation on cough above right scapula; bowels relaxed.
24th . .	100·5	100·3	99·2	100·2	100·7	...	...	...	Bowels relaxed.
25th . .	99·1	100·2	100·1	100·	100·2	...	...	...	... ...
26th . .	98·1	101·1	98·1	101·2	101·2	...	...	...	... ...
27th . .	98·3	100·2	100·	101·2	...	...	...	...	... ...
28th . .	...	...	98·4	99·4	101·	...	28	88	Bowels very relaxed.
29th . .	98·8	101·	99·2	102·4	102·4	101·8	32	120	Ditto.
30th . .	101·6	101·	100·2	99·6	102·4	101·2	28	104	Bowels not relaxed; skin cool at 10 a.m.
31st . .	101·2	101·6	98·4	101·8	101·	101·4	32	80	Ditto, 10 a.m.
June 1st . .	101·2	100·8	100·4	100·4	101·8	101·8	32	104	Low and prostrate; slight diarrhœa this morning; skin cool at 12.30 a.m.; skin hot at night.
2nd . .	100·4	102·	99·2	99·2	96·2	100·4	32	104	Pain under right nipple; bowels loose.
3rd . .	100·2	101·2	100·6	102·4	101·8	...	24	88	Bowels relaxed twice a day; light colour.
4th . .	100·	100·8	100·6	100·4	100·8	101·	...	...	Swelling of toes of left foot; no albumen in urine.
5th . .	99·6	100·	100·6	101·6	101·6	100·8	24	104	

After this a twenty-four hours' cycle was observed on June 13th and again on the 17th, of which the subjoined are the records:

First Day.

Hour.	Axillary temp.	Pulse.	Resp.	Meals.	Temp. of ward.	Notes.	Physical signs.
June 13, 1874							
8 p.m.	102·4	120, full	28	Half pint of milk	64·	Complained of sharp pain in right chest; skin dry	
9 „	102·6	120	32	...	„	...	
10 „	102·5	120	48	...	„	10. A linseed-meal poultice	
11 „	101·9	108	40	...	„	11. Sharp pain in right chest	
12 „	102·	104	34	...	„	Asleep; skin dry	
June 14							
1 a.m.	101·	96	40	...	„	Asleep	
2 „	100·8	72	40	...	„	„	
3 „	99·8	88,	36	...	„	„	
4 „	99·8	96	36	...	63·5	Awake	Right.—Dulness, upper half; dry cavernous sounds to fourth rib; croaking sounds below. Left.—Tubular sounds, upper fourth front; marked dulness, upper two thirds.
5 „	99·4	96	36	Cup of tea	63·	5.30. Bowels acted; motion not loose	
6 „	98·3	96	36	...	„	...	
7 „	99·8	84	40	...	„	...	
8 „	97·6	96	40,	...	„	Skin dry the whole night; cough slight; expectoration moderate. Respiration shallow	
9 „	98·	96	36	Bread, ham, tea	...	Skin cool	
10 „	97·6	112	32	...	...	Has just washed, &c.	
11 „	97	96	32	...	...	Awake	
12 „	96·	88	36	...	62½·	Skin warm, dry; urine 1 pint	
1 p.m.	97·6	88	28	Roast chicken, potato, champagne	...	„ „	
2 „	98·2	88	36	2 glasses of „	...	„ „	
3 „	98·4	104	36	...	64·	Skin hot; has been asleep	
4 „	99·8	112	36	...	...	„ awake; urine ½ pt., good colour	
5 „	101·	96	36	...	...	Asleep; skin warm	
6 „	102·	112	40	No eatables; half pint of tea	64·	„ skin hot and dry	
7 „	103·5	112	40	...	...	„	
8 „	103·4	128	36	...	...	Skin *burning* hot; nose very red; drowsy; has been asleep for three hours	

Second Day.

Hour.	Axillary temp.	Pulse.	Resp.	Meals.	Temp. of ward.	Notes.	Physical signs.
June 17, 1874						Weight 8 st. 4½ lb., having lost 1 st. 2½ lb.	
10 p.m.	101·8	108	32	...	67·	Awake; very quiet	
11 „	102·1	116	40	...	„	„	
12 „	102·1	108	36	...	„	Half asleep; skin moist; not coughing	
1 a.m.	101·	72	28	...	„	„ „ „	
2 „	101·2	100	44	...	„	„ „ „	
3 „	101·2	100	48	...	„	Awake; „ „	
4 „	101·5	100	40	...	66·	„ „ „	
5 „	101·2	112	28	5.10. Jelly	„	„ „ „	
6 „	100·5	120	40	...	...	„ „ „	
7 „	100·2	84	40	...	66·	Asleep; perspiring freely; „	
8 „	99·	96	32	8.30. Toast	„	Skin warm, not perspiring; urine passed twice in the night, dark and turbid	
9 „	98·8	84	32	...	...	Skin cool	
10 „	97·6	104	36	...	65½·	Skin very cool; awake, and washing himself	
11 „	98·6	104	32	...	„	Skin very moist	
12 „	98·2	120	32	...	...	Has been up to be weighed; exertion of getting into bed caused a little excitement, *vide* pulse	
1 p.m.	100·4	96	32	...	...	Asleep; skin warm	
2 „	100·8	96	32	...	...	„	
3.20 „	101·6	96	32	...	...	„ perspires a little	
4 „	102·2	112	48	4.45. Cold fowl and bread and tea	...	...	
5 „	102·8	120	40	...	...	Skin very hot; awake	
6 „	103·8	136	44	...	...	Skin burning hot and dry	
7 „	103·	112	48	...	...	...	
8 „	103·2	136	44	...	...	...	
9 „	102·	120	36	Bread and cheese and milk	...	Skin very hot	

Bowels not open.

June 22nd.—Seized with pain under right nipple on the morning of the 21st, which has continued ever since. Breath has been very difficult; patient has almost sunk from exhaustion; aspect pale and anxious; tongue red—furred in centre; pulse 120, resp. 48, temp. 99° (afternoon); crepitation, posterior right base; right side more resonant than left.

23rd.—Crepitation now audible over the whole right side, and cavernous sounds as above.

24th.—Feels relieved and ate a fair dinner (chicken); left leg swollen and œdematous; swelling in popliteal space tender on pressure, proceeding probably from plugging of veins.

Died on the 26th.

Summary.	At 8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.	10 p.m.	12 p.m
Averages . . .	99·1	99·7	100·4	100·3	100·9	101·1	101·4
No. of observations .	9	22	22	23	24	15	9

Autopsy twenty-four hours after death.

Both lungs somewhat collapsed. *Right lung* weighed 2 lbs. 11 oz., and was firmly adherent; the upper lobe appeared to be completely excavated, consisting of a mere bag of pleura; the lower and middle were consolidated with aggregations of miliary tubercle, in which centres of softening were visible; the middle lobe contained a small cavity of recent formation.

Left lung 2 lbs. 5 oz.; adherent; upper third appears to be consolidated with aggregations of miliary tubercle, caseating in parts, and showing a few small cavities, one the size of a walnut; lower lobe consolidated with ordinary red pneumonic hepatization. Its upper border contained a few grey aggregations.

Liver of nutmeg appearance, weight 4 lbs. 7 oz.

Intestines.—Mucous membrane of small intestine much injected. Solitary glands of ileum were caseous; some had

broken down and formed ulcers. The cæcum was one mass of ulceration.

The case only lasted six and a half months, and the post-mortem results may be summed up as—Excavations and recent tuberculisation of both lungs, with tuberculosis and ulceration of the intestines. The temperature course was highly pyrexial, but exhibited those periods of collapse which Lebert has so well described.

The thermometry of the "active third stage" shows greater extremes than that of any other, and includes both the highest and the lowest[1] temperatures, the actual maximum being 104·6°, the minimum 93·6°, a range of 11 degrees! The existence of these extremes, the marked pyrexia, and the subsequent collapse, show a close approximation to the thermal course, as far as we know it, of suppuration and of pyæmia.

Let us briefly summarise the results of the third stage active, as shown in Table IV (see page 96).

[1] Since this paper was read I have had under my care a case of this class where the temperature twice fell to 91·6°.

TABLE IV.—*Third Stage (Active).*

Hours.	104°.	103°.	102°.	101°.	100°.	100° and over.	99°.	98°.	97°.	96°.	95°.	94°.	93°.	Under 98°.	Maximum.	Mean.	Minimum.	No. of observations.	No. of cases.
8 a.m. . .	...	...	6	10	35	51	69	122	67	16	9	3	2	97	102·8	98·5	93·6	339	42
Percentages	...	...	...	...	10·32	15·34	20·35	36·	19·76	...	...	...	...	28·61	...	...	...	...	...
10 a.m. . .	...	...	2	7	13	22	26	11	10	7	...	...	...	17	102·7	99·2	96·5	7	76
Percentages	...	...	...	...	...	28·94	34·21	14·47	...	...	...	...	...	22·36	...	...	...	...	...
11 a.m. . .	1	4	12	45	69	131	77	92	26	9	5	2	...	42	104·	99·4	94·	342	38
Percentages	...	...	...	13·15	20·17	38·30	22·50	26·90	...	...	...	...	...	12·28	...	...	...	...	...
2 p.m. . .	...	14	33	78	79	204	84	42	12	3	...	1	...	58	103·8	100·2	94·8	346	39
Percentages	...	...	...	22·54	...	57·80	24·27	12·13	...	...	...	...	...	16·76	...	...	...	...	...
3 p.m. . .	1	5	8	7	9	30	4	1	...	1	1	1	...	3	104·6	100·6	94·	38	3
Percentages	...	...	...	...	...	79·	...	...	...	...	...	...	...	7·9	...	...	...	...	...
5 p.m. . .	1	10	43	79	98	231	86	32	6	1	1	...	...	8	104·1	100·4	95·6	357	40
Percentages	...	...	12·04	22·12	...	64·70	24·09	8·96	...	...	...	...	...	2·24	...	...	...	...	...
6 p.m. . .	...	...	...	8	6	14	2	...	...	...	...	...	...	...	101·3	100·9	98·4	16	2
Percentages																			
7 p.m. . .	1	4	11	9	11	36	5	...	...	...	...	...	...	...	103·9	101·5	...	41	2
Percentages	...	...	...	...	...	87·82	12·19	...	...	...	...	...	...	...	...	...	...	...	...
8 p.m. . .	...	20	47	85	110	262	70	29	9	4	...	...	...	13	103·6	100·5	96·	374	39
Percentages	...	...	...	22·72	29·11	70·05	18·71	7·78	...	...	...	...	...	3·47	...	...	...	...	...
9 p.m. . .	Too few.																		
Percentages																			
10 p.m. . .	...	2	16	33	15	66	3	1	...	1	...	...	...	2	102·5	101·2	96·2	71	6
Percentages	...	...	22·53	46·47	21·12	92·95	4·22	...	...	...	...	...	...	...	...	...	...	...	...
11 p.m. . .	...	...	2	5	...	7	12	...	...	1	...	...	...	...	101·4	100·1	96·8	...	...
Percentages																			
12 p.m. . .	...	2	11	10	16	39	10	3	...	...	...	...	...	...	102·8	100·6	98·4	52	5
Percentages	...	...	...	...	...	75·	19·23	5·76	...	...	...	...	...	...	...	...	...	...	...

The cases were 43, and observations were taken 13 times a day, viz., at 8, 10, 11 a.m.; 2, 3, 5, 6, 7, 8, 9, 10, 11, and 12 p.m., between 300 and 400 being taken at 8, 11, 2, 5 and 8. At 8 a.m., the majority of the temperatures are normal and subnormal; 36 per cent. were between 98° and 99°, and 28½ per cent. below 98°, the minimum being 93·6°, the maximum 102°, and the mean 99·5°; 20 per cent. were between 99° and 100°, and only 15 per cent. above 100°. In the few cases in which 100° was reached or exceeded, one was accompanied by acute ulceration of the larynx, another by rapid tuberculosis of the opposite lung. Even here a low morning temperature occurred, which was detected at 5 o'clock in the morning, but did not last until 8, the rise commencing early.

A third exceptional case was one where large hæmoptysis induced catarrhal pneumonia, and subsequently death. Thus are explained to some extent those isolated high temperatures among a great majority of normal or subnormal ones.

The rise, however, in this stage begins early, and at 10 o'clock 29 per cent. exceed 100°, 34 per cent. exceed 99°; while those under 98° are 22 per cent.

At 11, 38 per cent. are above 100°, 1 reaching 104°; 13 per cent. exceed 101°, while only 12 per cent. remain below 98°, the mean being 99·4°. The rise continues through the 2 p.m. records. Nearly 58 per cent. exceed 100°, 22½ per cent. 101°. The observations at 3 show a persistent rise: at 5 the percentage above 100° is 64, *i.e.* two thirds of the whole number, the maximum being 104·1°, the mean 100·4°; the minimum 95°. Only 2¼ per cent. fall below 98°.

The high temperatures are continued at 6 and 7, and at 8 p.m. we find 70 per cent. exceed 100°, and 22¾ per cent. exceed 101°, while only 3½ per cent. fall below 98°, the mean being 100·5°.

The notes at 10 p.m. show the highest temperature, but at the same time a tendency towards equalisation appears, 93 per cent. exceed 100°, the majority ranging between 99°

and 102°, only one observation falling below 99°. The mean is 101·2°.

The 11 and 12 p.m. notes indicate a subsidence, and to still more marked avoidance of extreme pyrexia or collapse. The mean at 12 is 100·6°.

The average maximum of the 43 patients was reached at 10 o'clock p.m.; but this is not invariably the case, for it may be arrived at at any hour between 5 and 10 p.m., and in isolated observations it has been reached even earlier, as will be seen in some of my twenty-four-hour cases.

This is of no great consequence, as the temperature having once risen, often remains for several hours pyrexial in character, but the point of importance is to ascertain the commencement of the rise and of the fall.

Before we quit the day temperature of this class, I venture to exhibit an interesting record of one of these cases taken thirteen times a day, from 8 a.m. to 8 p.m. for a period of eight days, which shows the general temperature course remarkably well, though the rise is somewhat earlier than in the majority of patients.

It was carefully carried out for me by Mr. M'Kinlay, on one of Dr. Symes Thompson's patients, who had a cavity in one lung and fresh tuberculization taking place in both lungs (see Plate II).

George B—, æt. 22, admitted into Richmond Ward under the care of Dr. Symes Thompson, January 14th, 1873.

History.—Cough commenced one year ago, with emaciation and large muco-purulent expectoration. Six months ago symptoms became worse, and occasional diarrhœa occurred. Cough worse at night, accompanied by dyspnœa; skin hot; night sweats considerable; appetite bad; tongue irritable; bowels regular; pulse 128; respirations 28.

Physical signs.—*L.* Cracked-pot sound and flattening to the fourth rib; dry cavernous sounds to the third rib; crepitation posteriorly, most marked at base.

R. Crepitation above the scapula, and from the clavicle to the second rib.

During the period these temperatures were taken, although, as will be seen by the chart, a considerable amount of pyrexia prevailed, he gained weight (see Plate II).

Plate IV shows pretty fully the temperature-course of this important class of cases.

The black dotted line (Plate IV, fig. 1, No. 1) gives the averages of the 43 patients, during the nine periods of the day already mentioned, viz., from 8 a.m. to 12 p.m. The thick black continuous line (No. 2) is a twenty-four hours' cycle of one of these cases, the same of which the eight-day series of temperatures have been exhibited in Plate II. The toothed line (No. 3) is an average of 7 similar cases taken hourly for twenty-four hours, and, like all averages, is not quite so striking as a single case.

A glance at these curves must convince the most sceptical that the pyrexia follows a definite order and law. The general form of the line representing the mean of a large number of daily observations tallies with similar periods in a twenty-four-hours' round of one of these cases; and if further proof were wanting of the close correspondence, it is found in the average of 7 other twenty-four-hour cases.

The rise and fall are well marked in all, though the maximum is not always reached at the same time. The black dotted curve (Plate IV, fig. 1, No. 1) reaches it at 10 p.m.: the toothed line (No. 3), or the average of 7 cases at 8 p.m., and the black continuous line (No. 2), at 6 p.m. When once reached, a fall, for the most part unbroken, takes place, extending through the night into the early hours of the morning, reaching 95° and even lower temperatures, and the effects of this we see in our low 8-o'clock observations, which are often normal or subnormal.

My colleague Dr. Symes Thompson, on my noticing this to him, suggested that these low temperatures might be owing to the axilla not being properly closed, and suggested my trying the mouth. I have done so in one twenty-four hours' case, and depicted it by the thin line (Plate IV, fig. 1, No. 4)). The general features of this curve are the same, the gradual ascent from the normal fall to the maximum of

101·9° (reached a little earlier than in the axilla cases) the rapid ascent with a few slight interruptions to the minimum 96·4° reached at 7 a.m., and then the recovery as the day advances. The general characteristics of "third stage active" temperatures may be summed up:—1st. Afternoon and evening pyrexia; 2nd. Rapid fall during night, and early morning; 3rd. Recovery in the later morning hours, and consequent normal temperatures.

The class of *third stage quiescent* or chronic cavity (Plate IV, fig. 2) is one of the commonest met with in practice, and has for its distinguishing thermometrical feature a remarkably low temperature course. The range of subnormal temperatures is very striking, and will be exhibited in the following case (Case 9), where cavities existed in both lungs, but no extension of disease took place, and some weight was gained. The temperatures ranged from 95·8° to 98·4°, and the mean temperatures are all below 98°, the 8 o'clock mean being 96·6°.

Case 9.—C. J— (Phthisis, III, with subnormal temperatures).

	8 a.m.	11 a.m.	2 p.m.	5 p.m.	8 p.m.	Physical signs.
March 18th .	96·4	97·2	97·8	97·6	98·	Cavernous sounds in both lungs.
„ 19th .	96·	97·8	97·2	97·	98·4	
„ 20th .	96·2	96·6	97·4	98·4	97·2	
„ 21st .	97·4	97·4	97·8	97·8	98·2	
„ 22nd .	96·8	98·2	96·8	98·2	97·6	
„ 23rd .	97·	97·2	97·6	98·4	97·6	
„ 24th .	96·8	97·	95·8	98·2	98·	
Average . .	96·6	97·3	97·2	97·9	97·6	

The observations on the "third stage quiescent" class were made on twenty patients, five and six times a day. About 150 are recorded at each period. Table V (on p. 101) shows an analysis of them.

Of the 8 a.m. temperatures only 6¾ per cent. reach 99°;

Table V.

Third Stage (Quiescent).

Hours.	102°.	101°.	100°.	100° and over.	99°.	98°.	97°.	96°.	95°.	94°.	Under 98°.	Maximum.	Mean.	Minimum.	No. of observations.	No. of cases.
8 a.m.	...	...	...	...	10	48	69	19	2	...	90	99·2	97·7	95·1	148	20
Percentages	...	...	...	...	6·75	32·43	47·	...	...	...	60·81	...	...	...	...	...
11 a.m.	...	...	1	1	33	70	29	10	...	...	39	100·	98·2	96·	143	18
Percentages	...	...	...	...	23·07	48·95	...	...	...	...	27·02	...	...	...	...	...
2 p.m.	1	...	2	3	46	62	33	11	1	...	45	102·	98·4	95·8	156	20
Percentages	...	...	...	...	29·48	39·74	21·15	...	...	...	28·84	...	...	...	...	...
5 p.m.	...	...	10	...	53	63	23	2	...	...	25	100·2	98·6	96·2	151	20
Percentages	...	...	6·62	...	35·10	41·06	15·23	...	...	...	16·55	...	...	...	...	...
8 p.m.	...	1	12	13	47	57	32	6	...	...	38	101·8	98·5	96·	155	20
Percentages	...	...	7·74	8·38	30·32	36·77	...	...	...	...	24·50	...	...	...	...	...
11 p.m.	...	1	1	2	14	19	4	2	1	...	7	100·	98·3	94·	43	6
Percentages	...	...	...	...	32·55	44·18	...	...	...	...	16·25	...	...	...	...	...

32½ per cent. reach 98°; while 60 per cent. fall below it; the minimum being 95°, the mean is 97·7°.

A few observations taken at 10 a.m. shows no marked change.

The 11 a.m. notes indicate a slight rise; 23 per cent. reached 99°, 49 per cent. 98°; 40 per cent. fell below it; the mean being 98·2°.

The 2 p.m. notes are slightly higher, and in a few exceptional cases exceed 100°; the mean is 98·4°.

At 5 p.m. the maximum is reached; 35 per cent. exceed 99°, and 16 per cent. fell below 98°, the mean being 98·6°.

After this there is a slight subsidence, and at 11 p.m. the mean is 98·3°. The black dotted line in the diagram (Plate IV, fig. 2, No. 1) represents the mean of twenty cases and shows the tendency of this class towards a subnormal character; the mean temperature of these twenty patients hardly reaching the normal 98·6.

The thick continuous line (No. 2) is that of the twenty-four-hours' cycle of a case in this category, which exhibits in addition to the above features a slight afternoon rise, somewhat resembling that of the active 3rd stage, though far less marked. As usually in phthisis, the morning fall is present.

Having thus reviewed the thermal features of these cases, as represented by the statistics, illustrative cases, tables and charts, two main facts stand out regarding consumption generally:

1st. The post-meridian character of the pyrexia when pyrexia exists at all.

2ndly. The remarkable fall at night and the subnormal temperatures of the early morning.[1]

[1] At the time when this paper was written I was not cognisant of Surgeon Nathaniel Alcock's (Army Medical Department) very able essay 'On the Nature and Varieties of Destructive Lung Disease, included under the head of Consumption, as seen among Soldiers,' which contains some excellent observations and deductions on the temperature of phthisis. He holds that the "earliest and most appreciable sign is an inability on the part of the person in whom the requirements for the development of tubercle exist, and are about to be set in motion, to maintain the temperature of the body up to the normal standard," and "that long after the deposit has taken place diminu-

The relations which these bear to a normal standard it is obviously of great importance to ascertain, especially whether or no the fall is peculiar to disease; and for this purpose I have consulted various authorities in order to arrive at that difficult problem, viz. the diurnal thermic course in a healthy man.[1]

Dr. Wm. Ogle's[2] careful experiments give some clue, but from ranges of hours and not exact periods being given they are hardly suitable for a chart. I have availed myself of Dr. Parkes' standard,[3] agreeing in the main with Dr. Ogle's, and used for testing the effects of

tion of temperature below the normal standard is frequently present during the intervals of local quiescence." He gives several well-marked cases of low temperatures in phthisis, and in some, readings of 96° F. and 97° were very common.

My colleague Dr. Pollock made a number of observations on cases of phthisis about the same time that mine were being carried on, and his conclusions, as stated in his lectures at the Brompton Hospital ('Med. Times and Gazette,' July 25th, 1874) were that "in ordinary quiescent phthisis (stage not specified) the minimum temperature, 98° or 97°, is in the early morning (3 to 7), and the higher temperature occurs from about 3 to 7 in the afternoon."

[1] Dr. Jürgensen, of Keil, has made a number of hourly observations on the day and night temperatures of healthy men, extending over several days. These give a chart slightly differing from the annexed one (Plate IV, fig. 2, No. 3), which I prefer to retain, because it is as well in comparing standards to aim at as nearly similar conditions as possible, and the fact of Mr. Kelly being under the same atmospheric conditions as the consumptive patients, and like Dr. Parkes' soldier, of the same nationality, renders the experiment more complete.

Dr. Jürgensen made some interesting experiments of the effects of food and starvation on normal temperatures. The experiment of starvation was carried out ruthlessly, for the man was not allowed even water for thirty-three hours. Slight diminution of the temperature, varying in amount from ·15 Centig. to ·2 Centig. followed, but the general course and fluctuations were maintained. On food being largely taken the temperature rose to the normal ('Die Korpe Wärme des gesunden Menschen,' 1873).

Dr. Finlayson ('Glasgow Medical Journal,' February, 1869) made a number of hourly observations on healthy children, but the temperature course in these differs so much from that of adults as to be useless for the purposes of this paper.

[2] 'St. George's Hospital Reports,' vol. i.

[3] 'Proceedings of Royal Society,' 1872.

exercise and alcohol on the body. The observations were carried out on a healthy young soldier, aged 30, of powerful muscular frame, and were taken every two hours from 8 a.m. to 8 p.m.; the figures representing the average of six days. Dr. Parkes' observations extend no further than the day, and being at a loss for a standard of healthy night temperatures, with which to compare the subnormal ones of phthisis, I requested Mr. Kelly, a clinical assistant at the Brompton Hospital, and a remarkably robust young man, aged 22, to submit himself to experiment. He kindly consented, and was therefore put in a bed in a private ward in the hospital, under the same equable temperature conditions as the patients, at 8 p.m., and remained there till 8 a.m. on the following morning, his axilla temperature being hourly observed by Messrs. Parry and Bernays. Beyond a glass of beer at 12 p.m. and some milk at 8 a.m. he had no food and slept soundly a great part of the night, notwithstanding temperature observations being regularly taken. The heat of the ward varied from 62° to 65°, and he perspired a good deal. Mr. Kelly's night temperatures, combined with Parkes' day records, are shown in the chart (Plate IV, fig. 2, No. 3). It proves that no special fall occurs at night, and therefore we may conclude that the fall in phthisis is abnormal and probably characteristic of consumptive disease.

To what is it due?

I first thought that it might be owing to night sweats, but on carefully consulting my night records, I detected signs of the fall where the skin continued hot and dry, and no perspiration existed. Again, profuse sweats occurred in the afternoon and evening in some instances, while the temperature remained the same. On the whole, there is a greater lowering in patients with night sweats than in those without this symptom, but as I am still pursuing my investigations on this point I will refrain from expressing any decided opinion.

Sleep does not appear to have much effect, as the temperature will sink to 95° or 96°, even if the patient remains awake, and as regards the effect of want of food and stimu-

lants, experiments of feeding these patients at night are not yet complete.

In all cases of phthisis two principal agencies appear to determine the temperature course:

1st. An excessive action of the natural processes by which the body heat is maintained.

2nd. The influence of collapse proceeding from the well-marked weakening of the constitutional powers in phthisis.

These two agencies are continually struggling for the mastery, and the result of this conflict is the temperature course of the disease. The influence of the first is seen in the rise in the afternoon and evening, well marked in the active forms of all three stages, and regularly recurring day after day, for long periods; the influence of the second is shown in the rapid nocturnal fall and the low temperatures of early morning; the collapse influence is also seen in the subnormal day temperatures occasionally occurring in all stages of the disease, and even where the active processes of lung tuberculization, of softening, and of excavation, may be taking place. It is, however, chiefly noted in the quiescent forms of the first and third stages.

When low temperatures accompany lung tuberculization or excavation, it is probable that the *collapse* influence is greater than the *pyrexial,* and therefore masks it.

Where the chart shows occasional fitful variations, these agencies are, perhaps, evenly balanced, and may alternately prevail, as is witnessed in the end-temperatures of consumption; and to these deviations noticed in advanced cases may be ascribed the prevailing and erroneous opinion that phthisis has no definite temperature course.

Dr. Burdon Sanderson mentioned to me an experiment which bears on this subject. He inoculated a guinea-pig and a dog with an equal quantity of purulent liquid. The temperature observations were then taken on both animals, and showed a lowering of temperature in the guinea-pig, and a considerable rise in the dog; the poison, therefore, produced pyrexia in the stronger animal, and collapse in the weaker one.

Table VI.

Weights.	First stage (active).		First stage (quiescent).		Second stage.		Third stage (active).		Third stage (quiescent).		Totals.	Per cent.
	No.	* Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.		
Gained weight . .	15	60·	2	...	5	...	7	21·21	13	68·42	42	46·15
Lost	5	20·	3	...	4	...	24	72·73	4	21·05	40	43·96
Stationary . . .	5	20·	...	...	...	...	2	6·6	2	10·53	9	9·89
Unknown . . .	1	...	...	...	1	...	10	...	1	...	13	...
	26	...	5	...	10	...	43	...	20	...	104	...

* Percentages are calculated with the Unknowns excluded.

The *weights* of the greater number of these patients have been taken before and after the thermometric observations, and the following table gives the result, and will, I think, repay a study as bearing upon the influence of pyrexia on weight (see Table VI, p. 106).

The thirteen unknowns are excluded, in calculating the percentages, but it is doubtful whether they ought not to be included among the losers, as the reason that the weight was not taken was generally that the patients were too ill to leave their beds.

We see that out of 91, 42 gained weight, 40 lost, and 9 remained stationary.

Of the first stage, active, 60 per cent. gained, 20 per cent. were stationary, and 20 per cent. lost.

In the second stage half of the patients gained weight.

The greatest loss is, as might be expected, in the third stage, active, where 72 per cent. lost weight; but even here 31 per cent. gained, and when we consider the desperate character of some of the cases, and their collapsed condition, how pyrexia continued in many for weeks and months, it is remarkable that so large a number did not lose weight.

The largest number of gainers is in the "third stage quiescent," where the percentage is 68; 10 per cent. were stationary, and 21 per cent. lost, and considering the number of double cavity cases this is noteworthy.

The reason of the large proportion of gain lies in the amount of food taken, combined with rest and comfort.

The quantity of food eaten by even those patients whose appetites are capricious is very considerable, owing to the regular stuffing system of the Brompton Hospital, and the number of delicacies supplied to meet every fancy. The scales often discover a gain in the weight of patients in whom it could have least been predicted.

The deductions from this table are—

1st. That, though the tendency of the disease is towards emaciation, gain of weight is possible in every stage, provided there be no diarrhœa or hæmoptysis, and that food can be taken and assimilated.

2nd. That pyrexia is not incompatible with gain of weight provided the appetite be good.

3rd. That while active third-stage cases are the least likely to gain weight, chronic cavity patients are the most likely to increase in flesh.

4th. That neither tuberculization nor softening preclude gain of weight.

These conclusions will be found in direct opposition to Niemeyer,[1] who states that he made out by "numberless examinations, with a thermometer and the weighing machine, that the decrease and increase in the weight of phthisical patients are respectively in relation to the height of the fever or to its disappearance;" but they confirm Dr. Ringer's[2] remark that "patients with a very considerable elevation of temperature, but who enjoy a good appetite do not lose flesh, provided always they are not employed in any active pursuit which entails much waste of tissue."

The influence of the various phthisical phenomena such as diarrhœa, hæmoptysis, pneumothorax, ulceration of the larynx and intestines, and albuminuria as also the effects of the contraction of cavities are postponed for a future communication.

[1] 'Text Book of Practical Medicine.'

[2] Op. cit.

DESCRIPTION OF PLATES II, III, AND IV.

PLATE II.

Case of George B—, aged 22. Temperature chart of his case, being a record of temperature taken thirteen times a day, from 8 a.m. to 8 p.m., for a period of eight days (see page 98).

PLATE III.

FIG. 1.—Phthisis, Stage I, active.

No. 1 (black dotted line).—Averages of 26 patients during seven periods of the day from 8 a.m. to 12 p.m. (see page 81).

No. 2 (thick black continuous line).—Twenty-four hours' cycle of one case (see page 81).

FIG. 2.—Stage I, quiescent, and Stage II.

No. 1 (black dotted line).—Average of five cases of Stage I, quiescent (see page 82).

No. 2 (thick black continuous line).—Twenty-four hours' cycle of one case (see page 83).

No. 3 (open crossed dotted line).—Average of ten II Stage cases (see pages 83, 87).

PLATE IV.

FIG. 1.—"Stage III, active."

No. 1 (black dotted line).—Averages of 43 patients during nine periods of the day from 8 a.m. to 12 p.m. (see pages 89, 99).

No. 2 (thick black continuous line).—Twenty-four hours' cycle of one of the 43 cases [the case of George B—, depicted also in Plate II], (see page 99).

No. 3 (toothed line).—Average of twenty-four hours' cycle of seven similar cases (see page 99).

No. 4 (thin continuous line).—Single case of temperature taken in the mouth (see page 100).

FIG. 2.—"Stage III, quiescent" and "normal" (page 100).

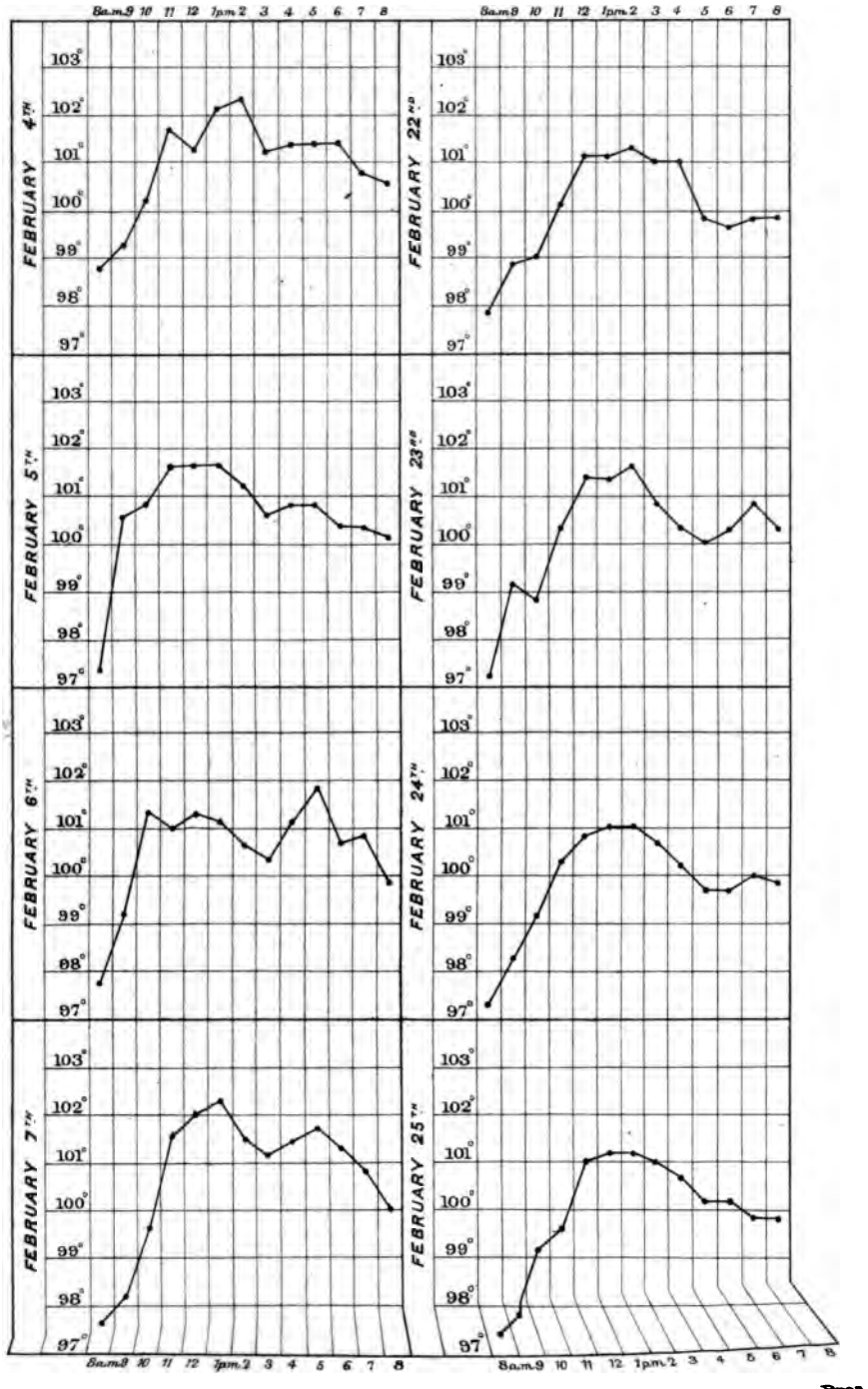
No. 1 (black dotted line).—Average of 20 cases taken eight times a day (see page 102).

No. 2 (thick black continuous line).—Twenty-four hours' cycle of one case (see page 102).

No. 3 (thin continuous line).—Twenty-four hours' cycle of normal temperature, (see page 104).

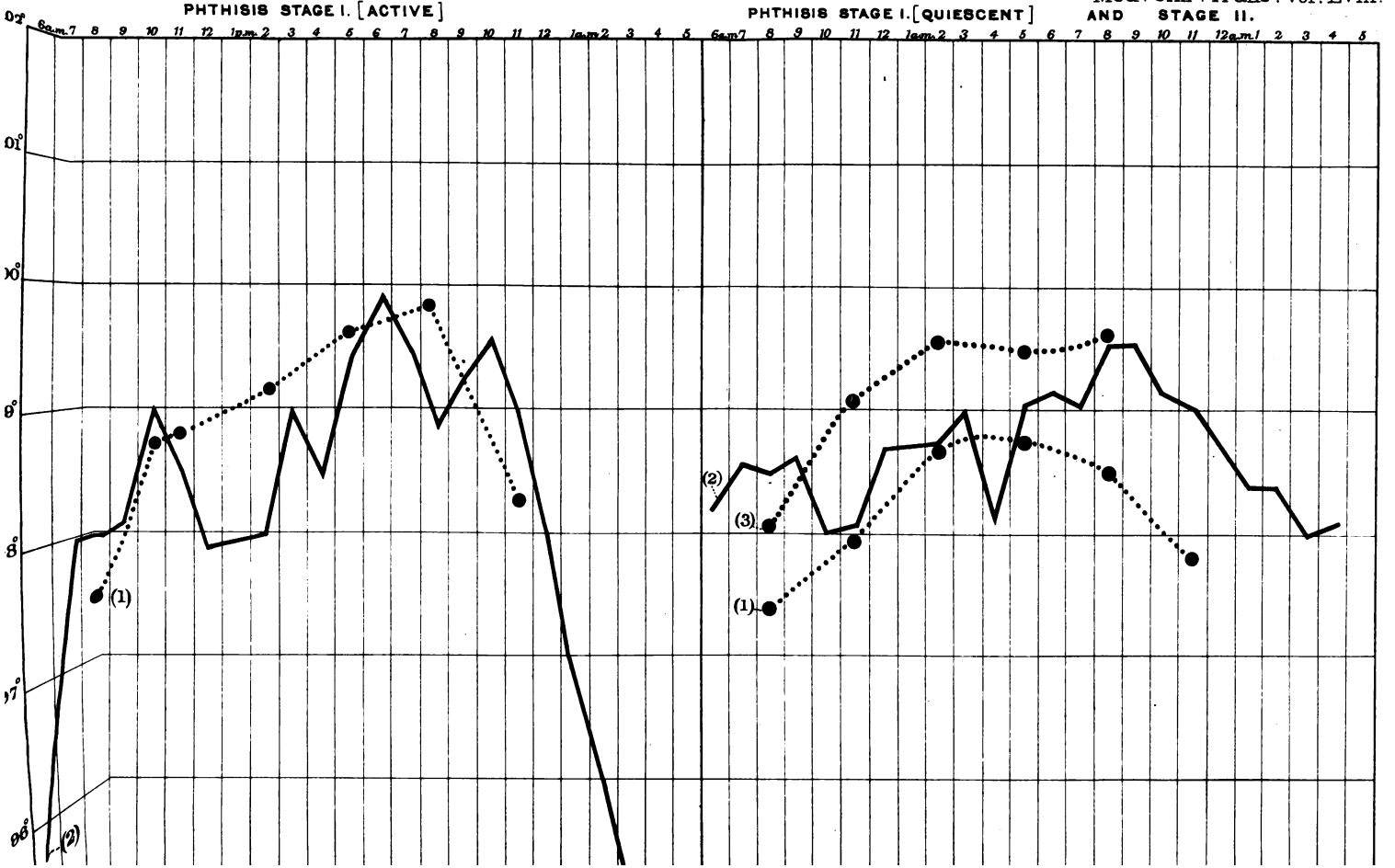
GEORGE B..... AGE 22.

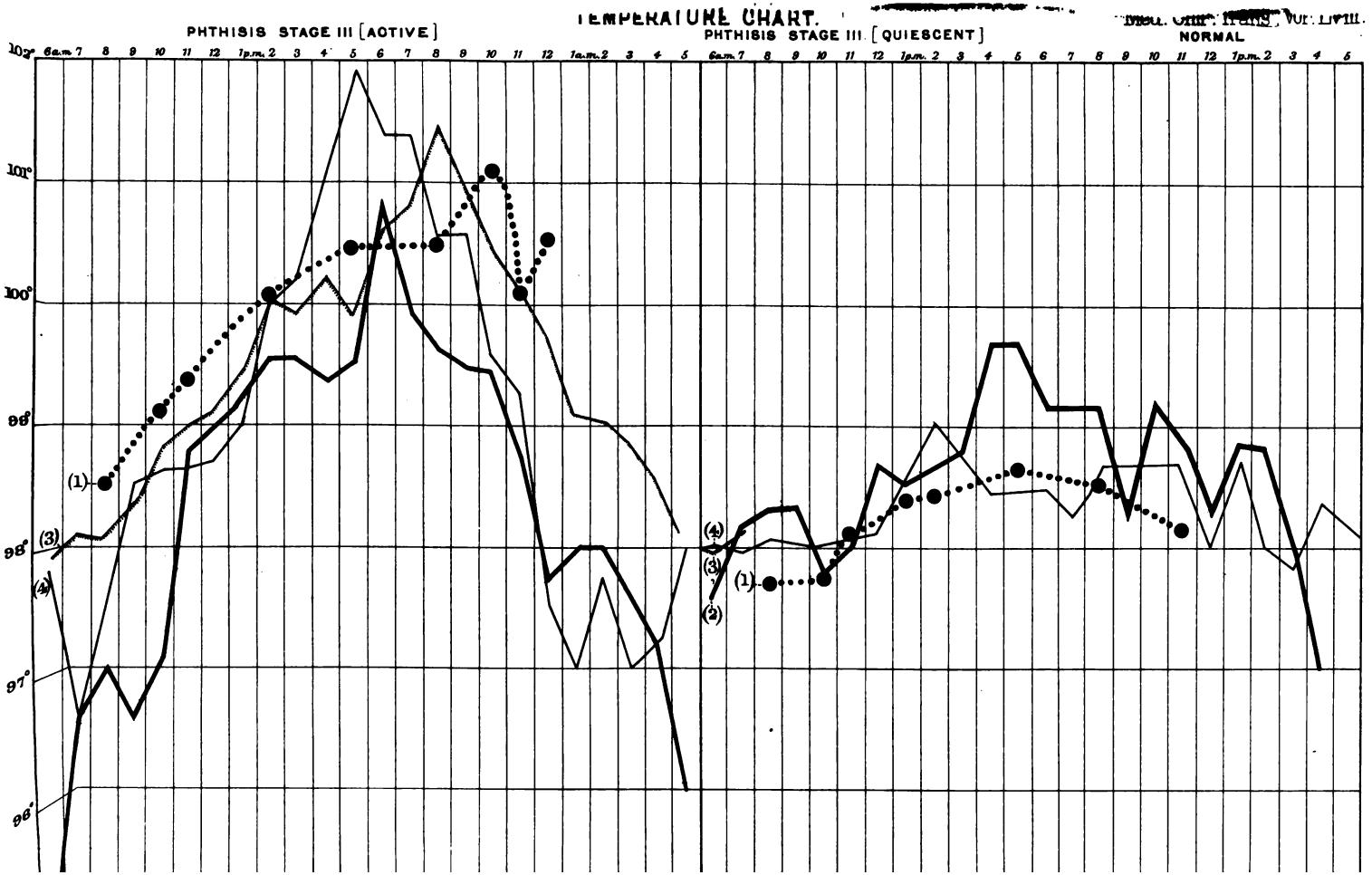
TEMPERATURE TAKEN IN THE AXILLA.



Mintern Bros. ?

TEMPERATURE CHART.


PHTHISIS STAGE I. [ACTIVE]
PHTHISIS STAGE I. [QUIESCENT] AND STAGE II.
6a.m. 7 8 9 10 11 12 1p.m. 2 3 4 5 6 7 8 9 10 11 12 1a.m. 2 3 4 5
6a.m. 7 8 9 10 11 12 1a.m. 2 3 4 5 6 7 8 9 10 11 12 a.m. 1 2 3 4 5
(1)
(2)
(3)


TEMPERATURE CHART.
PHTHISIS STAGE III [ACTIVE]
PHTHISIS STAGE III [QUIESCENT]
NORMAL
102°
101°
100°
99°
98°
97°
96°
6a.m. 7 8 9 10 11 12 1p.m. 2 3 4 5 6 7 8 9 10 11 12 1a.m. 2 3 4 5
6a.m. 7 8 9 10 11 12 1p.m. 2 3 4 5 6 7 8 9 10 11 12 1p.m. 2 3 4 5
(1)
(2)
(3)
(4)

CONTRIBUTION

TO THE

HISTORY OF LARYNGEAL PHTHISIS.

BY

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PART I.—A Short Notice of the History of Laryngeal Phthisis.

Previously to entering upon my own observations of this disease, I beg leave to retrace shortly the main features of the history of laryngeal phthisis, premising that after searching through various medical periodicals for a series of cases of laryngeal phthisis, carefully examined and reported in a practically useful form, my inquiries have been fruitless. This probably results from the fact that the present affection has not been universally acknowledged as a specific disease, and has therefore attracted, at all events in England, less attention than it appears to me to deserve.

It may also be added that owing to the comparatively recent discovery of the laryngoscope we have hardly yet had time for reaping its full usefulness in the study of laryngeal phthisis. It was only in 1857, during the summer months,

that Türck, of Vienna, endeavoured to employ the laryngeal mirror in the wards of the general hospital of that town. He was not successful, however, at first, and at the end of the autumn appears to have abandoned his fruitless attempt. In the month of November of the same year (1857) Professor Czermak, of Pesth, borrowed Dr. Türck's little mirror and overcame all difficulties; thus it was that Czermak created the art of laryngoscopy.[1]

The 'Dictionnaire Encyclopédique des Sciences Médicales' (1868), under the heading of *Larynx*, contains a full and interesting monograph of laryngeal phthisis by Doctors Krishaber and Peter.

The affection is defined as an ulcerous disease of the larynx of a tubercular nature, occurring during the progress of pulmonary phthisis, and occasionally preceding it. Its essential character is a tendency to destruction (of tissue) from a mere epithelial erosion of the mucous membrane to caries or necrosis of the cartilages.

This affection may be superficial or deep-seated; in the former case it assumes the features of a catarrh in which the epithelium undergoes destruction, leaving the mucous surface uncovered and secreting a large quantity of muco-pus. This muco-pus is in all probability the substance I have called white mucus or milky secretion[2] for want of a better name to give it, and wishing to defer calling it pus until I could obtain positive evidence as to its nature.

As the disease progresses it burrows into the tissues and affects the mucous glands, which become inflamed and are frequently destroyed, leaving an ulceration, even where no tubercles can be detected in the larynx. When the ulcerations have made their way through the mucous membrane, the cellular tissue, muscles, and cartilages become inflamed and necrosis sets in; the vocal cord may also ulcerate and even undergo complete destruction, when the whole laryngeal cavity is bathed in pus, mixed with portions of expectorations often tinged with streaks of blood.

[1] 'Mackenzie on the Use of the Laryngoscope.'

[2] 'Clinical Notes on Diseases of the Larynx.'

The above changes may take place without the formation of tubercles in the larynx, although tubercles are usually met with in that organ in the latter period of the disease; moreover, the ulcerations from the destruction of the mucous glands may become the seat of tubercular granulations. These tubercles assume either the form of granulations or that of ulcerations. When after death such granulations are incised they exhibit a grey tissue, nearly always yellow in the centre (their microscopical description is given); they are seated immediately under the epithelium by which they are covered. Soon afterwards the epithelium is shed, and the elementary constituents of the tubercle become atrophied and disorganized, being converted into a small indurated and ulcerated mass (*petite nodosité cratèriforme ulcérée*); occasionally the mass stands out, exhibiting an ulceration round its base. When several of the ulcerations are near to each other they join by extension of the tubercular formation. Thus originate larger ulcers usually superficial and with festooned margins. Their edges and centre are commonly the seat of small vegetations or vascular bodies, which, however, are very seldom developed to such an extent as to bear the name of polypus. At the same time the mucous membrane becomes vascular and covered with muco-pus. In conclusion, the anatomical changes met with in laryngeal phthisis may be divided into three stages. In the first the mucous membrane and its small glands are affected, although but superficially, a desquamation of the mucous membrane takes place, and an erosion or superficial ulceration occurs round the orifice of the ducts of these glands. The second stage is attended with destruction of the mucous membrane, causing considerable loss of substance; but the ulcerating process is also continued in depth and extends to all or nearly all the different parts of the larynx (muscles, cartilages, ligaments, and articulations), thus constituting the third stage.

After alluding to the laryngoscopic appearances of the larynx, the authors proceed to describe the symptoms of the disease. With respect to the voice, it is more or less changed

according to the stage of the pulmonary affection in which the laryngeal disease has supervened. If the larynx is affected at an early period of the pulmonary mischief the voice is but little altered, but if it begins at a later stage of the disease of the lungs the changes in the voice—hoarseness and aphonia, are most marked. Whenever, in the course of pulmonary phthisis, aphonia sets in rapidly, it may be concluded that the laryngeal disease is likely to be of the severest kind.

Aphonia may be cured, the laryngeal disease proving a mere simple catarrh of the mucous membrane, as happens in simple laryngitis, and quite independent of consumption; consequently laryngitis in phthisis should not always be considered as incurable. The cough in laryngeal phthisis has a peculiar sound, due to the fact that the larynx cannot close completely in the act of coughing, and a certain quantity of air passes between the vocal cords instead of its causing them to vibrate by means of a series of succussions as in ordinary coughing.

The expectoration has, of course, a double origin, being derived from both the larynx and the bronchial tubes. It is very difficult, if not impossible, to ascertain which portion of the expectoration is to be ascribed to either one or the other of these two sources.

Hæmorrhages from the larynx are very rare.

Pain is not often met with, and ulcerations of the laryngeal cavity are painless. When, however, the disease affects the epiglottis, then the throat begins to feel painful. There are, however, exceptional cases of pain in the larynx, these being met with where the laryngeal and the pulmonary disease run a rapid parallel course.

Deglutition, according to our authors' observations, was a painful act in at least one quarter of their cases of laryngeal phthisis, and in nearly all of them proved troublesome. The pain experienced while swallowing is occasioned by the pressure on the larynx exerted by the constrictors of the pharynx, as they contract while the larynx is raised in the act of swallowing. The dysphagia is sometimes such that the efforts to swallow bring on vomiting.

As to the nature of the disease, MM. Krishaber and Peter are convinced that it is invariably connected with pulmonary tubercles. They discuss Cruveilhier's statements, who, in 1834, did not believe the tubercular diathesis to be the only cause of laryngeal phthisis; he considered that chronic laryngitis neglected or unskilfully treated might become ulcerous, and that laryngeal phthisis could exist by itself independently of any pulmonary affection.

With respect to the diagnosis there are three points to determine—1st, is laryngitis present? 2nd, is the laryngitis ulcerous? 3rd, the ulcerous laryngitis, is it tubercular? The two first questions are readily answered by laryngoscopic examination; the last by an inquiry into the state of the chest.

If the larynx be affected, although without any sign of mischief in the lungs, the affection is perhaps what the authors call initial or preceding the pulmonary disease, or it may be simple chronic laryngitis or a syphilitic inflammation. Should unmistakable signs of phthisis be found in the lungs the laryngeal disease is not on that account to be considered necessarily as tubercular, it may be simple chronic laryngitis, but the larynx will probably become ulcerated when the laryngeal mischief is tubercular. An ulcerated larynx may, however, result from a syphilitic affection, and in order to place the diagnosis beyond doubt, an inquiry is to be made into the history of the case so as to ascertain whether there be any syphilitic taint present. In the absence of any such predisposition there can be no doubt that the laryngeal disease is tubercular.

Our authors next proceed to examine nervous aphonia in phthisis, and conclude that it is independent of tubercular disease, merely resulting from the state of debility connected with the pulmonary affection; it is consequently curable.

The prognosis is always very serious. The initial form is that which is likely to last longest in the latent stage; in cases where a remission of the pulmonary disease takes place the consecutive or secondary form may also exhibit a latent progress, and even become arrested. The ultimate or late

form is the last blow given to a constitution shattered by pulmonary disease. In short, laryngeal phthisis, Messrs. Krishaber and Peter observe, may become modified, arrested, or even undergo temporary improvement, but is never cured.

As to the influence of the expectorations from their direct contact with the laryngeal mucous membrane, our authors consider it as merely favouring disease (*condition d'opportunité morbide*), although many distinguished writers, such as Silvius, Louis, Trousseau, Belloc, and others, believe the sputa to exert a morbid action.

Treatment. — That adopted by MM. Krishaber and Peter appears somewhat bold in its application, although carrying much weight with it from the authors' experience and standing. Holding the laryngeal mirror by the left hand, with the right they introduce into the larynx a piece of fused nitrate of silver fixed to a caustic holder, and cauterise *deeply* (authors' italics) the affected surface. This operation is perfectly painless and is remarkable in that respect, the more so that solutions of nitrate of silver, even weak, produce much pain when applied to the diseased larynx. After a few days a superficial sequestrum falls away, when a fresh cauterisation is effected, and so on until the ulcerated surface becomes modified, which occurs in the course of a few days. Thus, the ulcerating process is arrested, the pain in the larynx lessened, and deglutition becomes easier.

A narcotic solution may be introduced into the larynx having the following composition :

℞ Extracti Opii,
Extracti Belladonnæ aa. 50 centigrammes (gr. $7\frac{7}{10}$),
Aquæ Laurocerasi 28 grammes (℥j).

The result of this application, it is stated, is certain (constant) and immediate ; the means is truly a mere palliative, but it sometimes proves extremely useful, especially where it is necessary to relieve intense pain in the act of swallowing.

Inhalation of narcotic vapours is also beneficial ; towards that object an infusion is prepared with 2 grammes (31

grains) of belladonna or stramonium leaves in 200 grammes (7 ounces) of hot water; the water is kept at a temperature of 160° or 170° F. in a special kind of inhaler, and the patient inhales the vapour for some minutes several times a day.

The acute laryngeal pain may also be relieved by hot fomentations applied to the larynx externally.

Dr. Pollock ('Elements of Prognosis in Consumption,' 1865) observes that he has found 8·66 per cent. of the whole number of (phthisical) cases to have their larynx affected,[1] but of those who attained the duration of four years only (19 out of 300) 6·33 per cent. had symptoms of laryngeal irritation. The extension of the disease to the organ of voice, although a much less frequent complication than diarrhœa, is a bad prognostic indication.

The question relating to the frequency of laryngeal phthisis in consumptive individuals is of much interest, and I regret that my own data with respect to this subject should be hardly sufficiently correct to allow me to express a result in figures; I should be inclined, however, to agree with Dr. Pollock's statement. My observations would give, perhaps, a rather higher proportion of cases of laryngeal phthisis, but, on the other hand, a certain number of these cases were sent to me at the time by my colleagues at the Hospital for Consumption, who, knowing I was engaged on that subject and took a special interest in it, directed some of their own hospital patients suffering from the throat to come to me. I am extremely obliged to these gentlemen for having thus promoted my object.

Niemeyer, 1868 ('Traité de Pathologie Interne,' translated into French under his directions), under the heading *Tubercular ulceration of the larynx*, admits two forms of laryngeal phthisis; one he calls infiltrated tuberculosis, the other miliary tuberculosis. He concludes that the disease (laryngeal phthisis) is never met with, but when the lungs themselves are tubercular, and observes, "we do not doubt

[1] Louis, from post-mortem examinations, places this proportion at 20 per cent. (Watson).

that tuberculosis of the larynx even in its diffuse form is of the same nature as pulmonary tuberculosis itself."

The seat of the affection, he observes, is usually the mucous membrane covering the transverse arytenoidean (probably arytenoideus) muscle, but tubercular ulcers may also be found in other places, such as the posterior aspect of the epiglottis. On that spot may be seen occasionally, though but seldom, little masses about the size of a millet seed, grey, and rounded, later turning yellow, then softening and undergoing disaggregation, and leaving in their place little rounded ulcerations of the size of a grain of semolina or hemp seed (the primary tubercular ulcer of Rokitansky). The union of several of these ulcerations from subsequent deposits becomes the source of a loss of substance irregularly defined and exhibiting raised and lacerated margins (the secondary tubercular ulcer of Rokitansky). It happens much more frequently, that the mucous membrane of the part affected first assumes a yellow tinge in consequence of its infiltration by innumerable small cells; the tissue becomes gradually relaxed, and a loss of substance takes place, first superficial, and by degrees spreading considerably in extent and depth. It is not unfrequent that the destruction is continued from the posterior walls of the larynx to the vocal cords, when it may happen that the posterior insertion of those ligaments is destroyed or undergoes considerable loss of substance. The tubercular ulcers of the posterior wall of the epiglottis in some rare instances perforate the whole thickness of that organ, the margins of the epiglottis remaining usually unaltered, while in syphilitic ulcerations this is not the case.

Ossification of the cartilages often coincide with tuberculosis of the larynx, and ossified sequestra occasionally form. Tubercular ulcerations may also perforate the laryngeal cavity, causing a fistula and cutaneous emphysema.

Niemeyer considers hoarseness in old-standing phthisis as a very probable sign of laryngeal phthisis, although it may also be due to muscular paralysis of the larynx without any organic change. The more the ulcerations approach the

vocal cord, the greater the hoarseness and more complete the aphonia. In other cases symptoms of hyperæsthesia are observed, characterised by great irritability and excessive cough and often terminating by nausea and vomiting; but, adds our author, "the symptoms we have just stated do not allow of the diagnosis of laryngeal tuberculosis so long as the existence of the same kind of affection in the lung cannot be determined; they can all of them be caused by other morbid changes in the larynx; it is therefore important, whenever a case of laryngeal disease is under consideration, to make a physical examination of the chest as carefully as possible, and put off expressing a positive opinion as to the tubercular nature of the affection, until the laryngeal symptoms can be connected with changes in the lungs as determined by percussion and auscultation.

"Cases of cure of laryngeal phthisis which have been reported, and the virtue of certain medicines called specifics in such instances are due in general to errors in the diagnosis. On the other hand, however, it is positive that a small number of recoveries have actually taken place."

On a few very rare occasions, œdema of the glottis supervenes, which proves fatal in a very short time.

With respect to the treatment, Niemeyer recommends the waters of Obersalzbrunnen or Kränchen of Ems, mixed with equal parts of warm milk. He advises the use of a strong solution of nitrate of silver applied to the larynx, and also frequent gargling with a solution of alum. The medicines, he adds, which prove most useful are narcotics; he prefers in this respect preparations of hyoscyamus and belladonna to those of opium. He also recommends living in a warm and slightly damp air, and in a place enjoying an equable temperature; he prohibits much talking and sometimes enjoins complete silence for weeks.

Niemeyer's description resembles that of MM. Krishaber and Peter in many respects, but all three authors appear to me too much to consider ulceration as the rule, as laryngeal phthisis may certainly exist for a considerable period without exhibiting any ulceration. Niemeyer, moreover, gives, I

think, too little attention to the state of the epiglottis, which is so frequently altered in its shape, and also to the intense pain in deglutition which accompanies the swollen condition of that organ. As to the topical use of solutions of nitrate of silver of which he appears to think well, I cannot refrain from observing that this means of treatment has never given me any satisfactory result. The local irritation this solution produces at the time is very great, and I much question whether any ultimate good follows.

Walshe ('Practical Treatise on Diseases of the Lungs,' 1871) observes that ulceration of the epiglottis gives rise to great dysphagia, especially of liquids, which frequently return by the nostrils. In proportion as ulceration destroys the vocal cords the voice degenerates into a hoarse whisper; he has never observed absolute aphonia; pain in the throat, stinging, pricking, or shooting, is more or less constant; if the epiglottis is free there is either no dysphagia or but very trifling difficulty in swallowing.

The physical signs are rough coarse respiration in the larynx, and sonorous, sibilant, or thin gurgling rhonchus, according to the dryness or moisture of the diseased surfaces.

Sir Thomas Watson ('Lectures on the Principles and Practice of Physic,' p. 204, 1871, vol. ii) observes that he knows of no such disease as laryngeal phthisis existing by itself, meaning that *scrofulous* (author's italics) ulceration of the larynx and trachea occurs only when the lungs are affected with tubercles, although it is sometimes a very early symptom. He also suggests the question as to whether the affection can be influenced by the contact of the matter which is expectorated and its frequent passage over the mucous membrane; and to this he answers, "Probably the fact, that the little mucous glands, wherewith the membrane is provided, are most numerous in the posterior part of the trachea and bronchi, and that these glands are especially prone to ulcerate, furnishes the truer explanation." Watson remarks that Louis, among 180 patients who died non-tubercular, only met once with ulceration of the larynx, while, of those who died of consumption, about one in every

five had ulceration of the epiglottis or larynx, and nearly one in three had ulceration of the windpipe.

Drs. C. J. B. and Ch. T. Williams, in their treatise on consumption, 1871 (p. 373), observe that some of the worse symptoms of the disease (phthisis) are those which indicate the various stages of ulceration of the larynx—pricking pain in the region, difficulty of swallowing, owing to the swollen or ulcerated epiglottis, hoarseness, gradually amounting to aphonia, and from time to time convulsive dyspnœa. These gentlemen have known of great comfort being given by the use of sulphurous acid in the form of spray, either alone or diluted with an equal amount of water. They state that Dr. Powell finds that a tincture of morphia, chlorate of potash, glycerine and soap, soothes the swollen epiglottis and renders swallowing more easy; but the authors conclude by remarking that little progress has been made in the treatment of the disease, and by hoping that the efforts of practitioners will some day be more successful than they are at present.

PART II.—Contribution to the History of Laryngeal Phthisis.

So much has been written upon the larynx in phthisis that there appears, at first sight, but little to add to it. It is only, however, since the comparatively recent discovery of the laryngoscope, and its introduction into general use, that the condition of the larynx, in cases of consumption, could be accurately investigated. Under the impression that there was still left a field for laryngoscopic inquiry into the present subject, I have submitted to a careful examination a fair number of cases of consumption attended with laryngeal disease; and these observations, together with the results they have yielded, form the ground-work of the present communication.

An inquiry of this kind is so far satisfactory that the pathological changes in progress can be seen at any

time, and their various stages investigated with the eye. The exploration of the chest is certainly the best means within our reach of ascertaining the condition of the lungs at any given time; but we cannot see into the lungs, and our hearing, educated by practice, must supply this want. We can now, however, by looking, satisfy ourselves of the state of the vocal cords, false cords, and epiglottis, together with the working of the laryngeal muscles. We can, moreover, by direct applications to the larynx, relieve the intense laryngeal pain which occasionally accompanies consumption; and we are able, I believe, to retard the progress of the disease in the throat, and in some cases prolong life by enabling patients to swallow where, from excessive pain and swelling of the part, deglutition is no longer possible. In cases of partial or total aphonia the state of the voice may be improved, although this satisfactory result is, as a rule, not easy to obtain.

Laryngeal phthisis is both a symptom and an extension of a pre-existing disease. It is a symptom, because, when clearly established by laryngoscopic examination, there is no doubt that the lungs either are, or will become tubercular if not obviously so at the time; it is an extension of a disease because, as a rule (I dare not yet say it is an invariable one, although it may be so), the pulmonary tissue is the primary seat of tubercular growth, mischief appearing subsequently in the larynx.

My connection with the Brompton Hospital gave me a wide field for the investigation of laryngeal phthisis, and I gladly availed myself of it. Consumptive patients, with throats in every conceivable stage of disease, applied for treatment in the out-patients' room of the hospital: some I examined on the spot; when pressed for time others were appointed to call at my house, where I inquired into their cases. Notes were taken carefully while the examination of the patient was in progress.

Predisposition to Laryngeal Phthisis.

There is, apparently, according to my experience, a very great difference between the predisposition to laryngeal phthisis in hospital and private practice; it is much greater in the former than in the latter case. I mean, that, in a given number of cases of consumption, the larynx, I believe, will be attacked much more frequently among those who attend hospitals than others; moreover, as a rule, the laryngeal disease will run a more severe course amongst hospital patients. I was prepared to find, by inquiring into the occupations of the patients I attended at the Brompton Hospital, that those who made most use of their voice, such as hawkers, would be most liable to laryngeal phthisis; but on examining into the subject this turned out to be hardly the case. The trades or employments were recorded in fifty-eight cases of laryngeal phthisis; most of the patients were engaged upon indoor labour, including shopmen, domestic servants, and clerks; there were two cases of butchers' assistants whose duty it was to call out the weights of the meat as it was taken off the scale, which appears to be an occupation very laborious for the vocal organ. There were one mattress maker, four needlewomen, including one machinist, one naval captain, one reader to the press, and only one labourer. I cannot find any case of a hawker recorded.

Of 309 phthisical patients I attended at the hospital in 1869, none of whom were recorded as suffering in any way from the larynx and who may be safely considered as free from laryngeal disease, about eighty were engaged in sedentary or indoor work, giving a proportion of 26 per cent.; while, out of the 58 cases of laryngeal phthisis, about 30 patients were employed on sedentary or indoor labour, yielding a proportion of about 52 per cent. of the total number. There appears to be consequently, a decided predisposition to laryngeal phthisis amongst those engaged upon indoor work, and who failed, therefore, as a rule, to take a sufficient amount of exercise

in the open air, and whose breathing was carried on in an ill-ventilated atmosphere.

I could not clearly trace the affection to overwork of the vocal organ, the two cases of the butchers' assistants calling out the weights are, however, interesting to record, the more so that out of the 309 phthisical patients without any laryngeal complication there does not occur one butcher. The case of the reader in a printing-office is also worth noticing.

From the above statement it appears that deficient exercise and want of pure air, and, I might add, neglect of hygiene, with which is often united mental anxiety, are the main predisposing causes of laryngeal phthisis. This explains how it is that those consumptive patients who can live in healthy districts, take exercise in the open air, procure good wholesome food, enjoying certain comforts, and freedom from anxiety, are less liable than others to suffer from an extension of the disease to the throat. In fact, laryngeal phthisis is apparently a low form of consumption, or a feature of phthisis occurring in a debilitated individual; hence the importance of attending as much to the general state of health in these cases as to the local mischief.

The task I set myself to work out was to examine together the physical signs of the chest and the state of the larynx where affected, in order to ascertain whether the progress of the disease could remain limited to either of these organs, or whether a change in one part was attended or not with some similar change in the other. I also endeavoured to trace the local alterations occurring in laryngeal phthisis as the disease ran its course, by following up such cases for a considerable length of time; and, finally, every possible attention was given to the treatment.

Before entering strictly upon the subject of laryngeal phthisis, I must beg leave to begin by drawing attention to that state of the larynx, which is occasionally met with in consumption, characterised merely by weakness of the voice, and sometimes aphonia, without there being any inflammation or

tubercular growth in the vocal organ. The patient finds out that after speaking for a length of time the voice drops, or it fails; while singing, reading aloud, or teaching has to be discontinued, and the act of phonation becomes an effort. This affection may remain stationary for months, quite independently of the course taken by the pulmonary disease; in some cases it becomes worse; by degrees speaking may require a considerable effort, when the sound of the voice assumes a high and unnatural pitch, but at that stage the patient finds it so laborious to utter sounds that words are usually spoken in a whisper. A laryngoscopic examination shows at first sight no apparent mischief, but on the patient attempting phonation it will be seen that the vocal cords do not move freely. In most cases the tension of the cords is observed at fault, while their adduction is unimpaired; in a few others the adduction is incomplete. Where tension is deficient, the larynx, instead of exhibiting long straight vocal bands occupying the whole antero-posterior diameter of the larynx, as in the normal state, shows them to be shortened and somewhat shrivelled, the whole larynx contracting round its centre in its attempt to produce a sound; while sometimes the cords entirely disappear from view.

In the other case the length of the cords does not appear altered, but they fail to approach each other sufficiently to vibrate.

This last state of the laryngeal muscles is usually met with in hysterical aphonia, and, indeed, it appears to exhibit in consumption the peculiar intermittent character it is found to have in hysteria, the voice leaving and returning from time to time without any apparent cause. Still the present laryngeal affection may be met with in phthisical male patients who can in no way be considered hysterical.

Now, although these two functional changes in the larynx might appear to have different origins, the tensors or crico-thyroid muscles being supplied exclusively by the superior laryngeal nerve, while all the other muscles are supplied by the inferior laryngeal, still both nerves arise from the pneumogastric which is also distributed to the lungs,

and they all three meet in the ganglion on the root of the track of the pneumogastric. Therefore it appears to me possible that the deficient state of nutrition or abnormal condition of the lungs should also weaken the functions of the laryngeal nerves, by a reflex action through the ganglion. In hysterical aphonia the mischief appears more local, and by faradisation of the laryngeal muscles a complete cure can often be affected; while in phthisical aphonia without organic change, electricity, although it may be of use, often fails to restore the voice or even to improve it materially. On the other hand, in these cases, an improvement in the state of the lungs and the general health will often be attended with a simultaneous favorable change in that of the voice.

Trousseau ('Clinical Med.' vol. iii) alludes to this particular form of aphonia in consumption, unattended by any organic change. He observes ". . . . but likewise and without any material lesion, as is shown by examination with the laryngoscope, pulmonary tuberculisation sometimes produces nervous aphonia." This affection is also noticed, as stated in the first part of this paper, by MM. Krishaber and Peter.

The following is a case illustrating the usual form of nervous aphonia in phthisis, that which is due to want of power of the *tensor* muscles of the vocal cords.

(Case 46.)—Mary Ann T—, middle-aged, pregnant (and has a large family), consulted me on the 25th June, 1869. The physical signs of the chest were—on the left side in front respiratory murmur, with humid crepitus just below clavicle, falling in at right apex with some dulness, and deficient murmur on that same side, abundant mucus crepitus and tubular breathing under the third rib. She had an attack of hæmoptysis, which lasted two days, two months before applying.

I examined her with the laryngoscope on the 23rd July; no organic change was to be seen in the larynx; the cords looked healthy. On attempts at phonation the arytenoid cartilages moved freely towards each other, showing a proper

adduction of the cords, but they were drawn out of the field of the mirror and appeared deficient in tension.

She lost her voice three months before coming to me, a word being, however, spoken out now and then. I understood her to say she had a previous attack of aphonia, from which she had recovered.

On applying faradisation to this patient's larynx I thought I observed a want of sensibility of the organ, as it was not conscious of a fairly powerful electric current, which, however, was felt when stronger. At first faradisation caused but a slight improvement, but she also took cod-liver oil and iron, and was eventually benefited considerably by the treatment. On the 21st September her voice was much improved, though still weak and hoarse.

In the following case it was not the tensor muscles, but the adductors, which were at fault.

CASE 47.—Henry G—, æt. 22, a labourer.

Physical signs.—Left and right side (in front) shade of dulness from apex to middle. Left in front crackling and sibilus, no obvious respiratory murmur. Posteriorly shade of dulness on both sides. Left supra-spinous fossa crackling and metallic sounds. Right posteriorly respiration bronchial with crepitus. Voice very weak and hoarse.

Laryngoscopic examination.—Throat so irritable that the mere introduction of the glass into the mouth caused spasmodic contractions. Recourse was had to a waterproof bag filled with a mixture of ice and salt tied round the patient's neck and in contact externally with the larynx; a few minutes later the examination proved quite easy. The laryngeal mucous membrane and that of the arytenoid bodies was rather relaxed and red; there was some congestion of the cords, epiglottis somewhat pendulous but during the act of coughing the larynx became plainly visible. It was then distinctly observed that on attempting phonation the vocal cords failed to approach each other, although both of them moved about half-way towards the median line. This patient had two attacks of pleurisy thirteen months before (August twelve

months), the second being rather a relapse, and he never quite recovered his health since then. There was nothing defective in his voice at that time, but just before the following Christmas he became hoarse and by degrees nearly lost his voice completely. In the following month of May he had a very bad cold, with an attack of hæmoptysis. He began to suffer from a fistula in ano about the time when his voice became affected.

I will now relate an interesting case, in which both the adductors and tensors failed in their action, there being extensive disease in the lungs.[1]

John F—, æt. 21 (Case 36), has been subject to aphonia for the last two months. His speech is now (19th March, 1869) delivered in a cracked tone of voice, and some words are dropped in a whisper; he has never suffered from hæmoptysis; one of his brothers died of consumption.

Physical signs of the chest.—At right apex in front, shade of dulness, cavernous respiration with crackling to third rib; at base respiration vesicular with some crepitus. On that side posteriorly unmistakable signs of cavity and consolidation. Left lung tolerably healthy.

With the laryngoscope the posterior halves of the vocal cords are seen to have lost their natural hue and acquired a red glistening appearance. The cords fail to stretch properly, as shown by the middle of the chink between them remaining open when a sound is attempted, while their ends come into mutual contact.

The arytenoid cartilages are observed to be drawn anteriorly, in the act of phonation, and to approximate so close to each other as to meet, or very nearly so, the cords being at the time much shortened. On abduction the cords do not move quite symmetrically, the left assuming more of a diagonal position than the right, they appear uneven. Supraglottic region red and relaxed.

The patient was treated with cod-liver oil, syrup of phosphate of iron with quinine, and croton oil liniment rubbed

[1] 'Clinical Notes on Diseases of the Larynx,' p. 104.

occasionally on the right side of the chest. Faradization was applied repeatedly to the vocal cords.

The progress of the case was quite satisfactory, and on July 16th both vocal cords moved freely and symmetrically in the act of phonation; they were not shortened and came tolerably well in contact throughout their whole length, showing a marked improvement in their power of tension. The arytenoid cartilages were no longer displaced in an anterior direction, and their lateral movement was quite free.

The patient could sing the scale without any difficulty, and the sound of his voice was much improved. I saw him next on the 8th July, when his voice might have been considered as quite natural.

From the interest of this case I may perhaps be allowed to reproduce it on the present occasion.

We may conclude that hoarseness, or aphonia, may result from phthisis, either directly or indirectly, without any organic affection of the larynx; and in these cases electricity may be productive of much benefit, and even bring on recovery, although the treatment can hardly be considered so successful as it is in simple hysterical aphonia.

I shall now enter more directly upon the subject of my communication, namely, *laryngeal phthisis;* but it is necessary that some explanation be first given as to the meaning this denomination is proposed to convey.

I propose to include under that name both what might be called *tubercular laryngitis* and *laryngeal phthisis*. The former would mean of course laryngitis or inflammation of the larynx connected with tubercles, the latter would mainly convey the idea of an adenoid growth (tubercular deposit) in the larynx independently of inflammation. It often happens that when inflammation accompanies laryngeal phthisis it possesses no peculiar appearance beyond what is observed in common laryngitis; but it differs in this respect—that while in common laryngitis it yields readily to treatment, in tubercular laryngitis it frequently goes from bad to worse, whatever be the mode of treatment adopted. There is, apparently, in

laryngitis accompanying phthisis, a local source of irritation acting incessantly and keeping up the inflammation, which is not the case with ordinary laryngitis. The usual signs of laryngeal inflammation may last for a considerable time in phthisis and sometimes merge into a permanent thickening without any tubercular granulations being visible. It is obvious to me, however, that the thickening is of a tubercular nature; the parts forming the vocal organ, which are naturally soft and flexible, become stiff and indurated, and on being punctured with a scarifier, convey the impression of a hard gritty body. Moreover, a white milky secretion (probably purulent), varying in thickness, is observed on the walls of the larynx, and sometimes oozing out between the arytenoid bodies, in nearly every case of inflammation of the larynx accompanying phthisis. It is so frequently met with under those circumstances, that its presence has enabled me to diagnose tubercular disease of the lungs where the pulmonary symptoms were, indeed, very questionable.

In many cases, however, perhaps in most, a granular appearance is seen in various parts of the larynx; it may be visible either on the arytenoid bodies or epiglottidean folds, the false cords or the epiglottis; the part looks as if speckled over with a number of small white dots the size of a pin's head or millet seed. The tissue in which these little masses are imbedded is red and congested, but if scarified the part feels resistent, and yields much less blood than would have been anticipated. It may happen that the granulations are concealed by the white secretion I have referred to, but if that be wiped off with a brush the state of the laryngeal mucous membrane becomes at once visible.

Where the epiglottis is thus affected it becomes very hard and altered in shape, assuming a shrivelled up and contracted appearance. The pain felt in the throat in these cases is sometimes excruciating, especially during the act of deglutition. I have seen patients dreading to swallow, throwing the head into every possible position, and applying the hands tightly against both ears with the vain hope of obtaining some relief during deglutition, while intense pain

was marked upon their features. When the epiglottis is much contracted it fails to protect effectually the orifice of the larynx, and the discomfort of liquid food not finding its way readily into the œsophagus is added to the pain. Those extreme cases are fortunately but seldom met with. I have, however, seen a sufficient nnmber of them to be thoroughly impressed with their serious and extremely painful character.

It sometimes happens towards the termination of the disease that growths or little tumours form on the larynx; they are seen, perhaps, mostly on the arytenoid bodies, but may be met with on other parts of the larynx, and are accompanied by softening and ulcerations of the laryngeal mucous membrane. These changes indicate, of course, a most unfavorable condition.

It is remarkable that, although in some instances the soft parts of the larynx should become so thoroughly indurated, still the vocal cords continue moving apart tolerably freely in the act of respiration, so that actual dyspnœa beginning at the larynx is seldom met with. The detection of the very first occurrence of softening of the laryngeal mucous membrane is not always easy to determine. When this change has set in, the white secretion becomes thicker, and on removing it with a camel's-hair brush the surface of the mucous membrane is seen to be pitted, or, if I may be allowed the comparison, somewhat like moth-eaten cloth; or the follicles on the posterior aspect of the larynx, lying between the arytenoid bodies appear enlarged and somewhat excavated at their base. In other cases positive ulceration is met with, the posterior ends of the vocal cords being not unfrequently involved in this change.

I have often attempted to ascertain whether the succession of changes the larynx undergoes follows a determined course; whether weakness of the vocal muscles is always the first change, next, irritation and inflammation of the part, then swelling and induration with a granular condition, and, finally, softening and ulceration.

I cannot say, however, that I have succeeded in doing

this, and question whether those changes invariably follow each other. I have observed, for instance, that a merely weakened condition of the muscles of the vocal cords may often exist throughout the whole course of a case of consumption without its passing into inflammation. Indeed, it is very seldom, in a case of confirmed phthisis, that the power of phonation is not more or less weakened; this may not be apparent, but the patient will be conscious of it. Tubercular disease in the larynx may, however, also commence with a want of power in the production of sounds, the earliest signs of inflammation being superadded weeks or months later. A weakened state of the voice is a sign of no little importance, especially when it has lasted for a considerable length of time. Patients are often aware of this, and it does not unfrequently happen that a lady with consumption in her family, although in apparently perfect health, suddenly finding her voice beginning to fail when singing, will at once anxiously seek for medical advice.

Suspicious symptoms may be heard in such cases at the apices of the lungs, such as some harshness, slight vocal resonance, and deficient respiratory murmur, without, however, any appreciable dulness on percussion. These cases may do well under treatment, the power of singing returning, although care should be taken by such patients to use the voice as little as possible for some time.

Inflammation of the larynx, with the presence of the white milky secretion, may be accepted as a very strong evidence of phthisis, although, of course, if unaccompanied by any physical signs of the chest, one should pause before giving a positive opinion. I have related a case ('Brit. Med. Journ.,' 24th October, 1874,—"Consumption, a form of septicæmia") of a gentleman in apparently perfect health, in whose throat I detected the presence of the white secretion alluded to above. There was no sign of mischief in the lungs beyond a slight dulness on percussion at the left apex; yet, twenty-six days afterwards, this patient was seized with hæmoptysis, which proved the beginning of acute phthisis, and he died a month later.

Laryngeal disease in phthisis leads to aphonia. Of course,

where the soft parts of the larynx are thickened and indurated, it is natural that the voice should be affected, but it is often remarkable how little laryngeal mischief may produce this result. In the early stage of the aphonia with thickening of the larynx, the voice may be restored by electricity, but we must not flatter ourselves that the recovery will be permanent. In an old-standing case of aphonia with chronic thickening, electricity will not be of much avail, although a few words, and perhaps some sentences, may be obtained by this means. I question whether ulceration of the larynx be necessarily attended with aphonia, unless the ulceration should be situated on or very near the vocal cords.

The prognosis derived from laryngeal phthisis is, I fear, as a rule, unfavorable. It is specially so when softening of the larynx has positively set in. Still, with proper management, especially respecting climate, I have seen patients with the soft parts of the larynx considerably thickened, and obliged to converse in a whisper, spend a very comfortable and pleasant winter in the south of France, and certainly be no worse in the spring than they were in the preceding autumn. As a rule, the condition of the throat remains chronic so long as the state of the lungs is quiescent, and, consequently, the pulmonary conditions and general health must be always taken into account, together with the state of the larynx, in reference to prognosis. I now have to remark that the results of my observations as to the state of the throat compared with that of the lungs, has led me to conclude that similar changes do not take place simultaneously in both organs. Thus, patients having large cavities in the lungs may continue for a considerable time with a thickening in the larynx, exhibiting no signs of softening; or there may be chronic irritation in the throat, with the secretion of white mucus, and no aphonia, remaining unchanged to the termination of the disease. I do not think that laryngeal consumption is cured any more than pulmonary consumption, and it is obvious enough that if the affection of the throat should be owing to tubercular or adenoid growth,

the cause of irritation cannot be removed. If, however, such a satisfactory result cannot be obtained, we can do a great deal towards relieving the irritation and changing its acute into a chronic form by removing the patient into a proper climate.

There is no contagious or especially morbid quality in the air expired by those who suffer from laryngeal phthisis, although it is frequently possessed of a very disagreeable fœtor. I have found, however, the breath of such persons extremely irritating to my own larynx, and a protracted laryngoscopic examination in cases of phthisis has frequently been productive in me of much discomfort and hoarseness. This influence of expired air when breathed insufficiently diluted, is not, I believe, limited to laryngeal phthisis, although, perhaps, the breath in this affection and, I might add, in consumption, is still more irritating than under other morbid conditions.

All work with the laryngoscope when carried on for a certain time has had an irritant effect on my own throat, so much so that the members of my family have frequently been led to infer, from the sound of my voice, that I had been much engaged with the laryngoscope on that particular day. At times my voice was very hoarse with a constant desire to clear the larynx, which proved extremely troublesome; wiping out my own larynx with a camel's hair brush only removed a small quantity of perfectly clear mucus. This continued for months till, at last, I had to adopt some means to put an end to it, and with this object in view I wore a charcoal respirator every time I had to do any hospital work with the laryngoscope. This plan was perfectly successful. The liability to laryngitis from breathing expired air remains, however, although I am not in the least subject to irritation in the larynx from other causes.

The foregoing remarks certainly show that a state of irritation of the larynx may proceed from breathing too close to the mouth of another person, and the effect will be the same in crowded and ill-ventilated rooms.

Although healthy expired air is irritating to breathe,

air expired from those who suffer from laryngeal phthisis, and also I might say from phthisis, unaccompanied by any laryngeal disease, appears to me, as previously stated, possessed of a still more irritating character. All substances present in the blood under a form that allows of their being eliminated in a volatile state, find their way out of the body through the lungs, so that expired air contains many substances besides carbonic acid. A well-known experiment of Claude Bernard, which I have frequently repeated, consists in the injection of an aqueous solution of sulphuretted hydrogen into the veins of a dog, and testing the air expired by the animal a few seconds later, when it is found to be highly impregnated with sulphuretted hydrogen. This shows how very readily volatile substances are given out of the blood through the substance of the lungs. I have also observed on one occasion a man who was wearing a tracheal canula, and whose breath, therefore, must have come from the lungs without passing through the mouth, expire air smelling strongly of onion or garlic. Now, some of these volatile substances appear to be irritating to mucous membranes; indeed, they are chiefly effete matters, and cannot therefore be taken into the body without producing mischief.

It is not improbable that the tendency to consumption resulting from a sedentary occupation in crowded and ill-ventilated rooms is partly owing to the irritating influence of expired air, but this tendency may be due also to some property possessed by expired air which may be allied to that of a poison, just as urea acts as a poison when injected into the blood, or when its elimination from the body is checked by disease of the kidneys. Now, when pure air is inhaled it becomes charged in the lungs with the volatile constituents of the blood which have diffused themselves through the pulmonary tissue; but if the air inspired be already loaded with volatile substances from other people's blood, these substances will diffuse themselves through the lungs of those who breathe them into their own blood, and act as a poison; and to the present substances must be added carbonic acid, which will also be absorbed for the

very same reason. No wonder then that the nutrition of lung-tissue charged with carbonic acid and volatile substances acting as a poison should become altered and eventually diseased.

Treatment.—The treatment of laryngeal phthisis may be classed under three heads—1st, local; 2nd, medicinal; 3rd, climatic; the second and third including means of treatment which apply, of course, to pulmonary phthisis as well as to the laryngeal disease.

The local treatment is clearly that which should be applied first. The use of solutions of nitrate of silver, perchloride of iron, or of any strongly astringent and irritating fluid, I have discarded. I do not find these applications do any good, and they produce much distress at the time. Solutions of bromide of potassium, and alum in glycerine, or gum water, will answer the purpose much better; when applied freely to the larynx with a camel's-hair brush, they often produce a temporary relief of the pain. I have, however, found a solution of iodine in olive oil introduced into the larynx to answer the purpose more effectually; ten to twenty grains of iodine with two or three grains of iodide of potassium dissolved in one ounce of olive oil forms a solution which keeps fairly well. In case of much irritation of the larynx a few drops of this solution may be mixed with an equal number of drops of olive oil before it is used; it is remarkable to what extent olive oil checks the irritating action of iodine, which it dissolves readily, and the operation is sometimes attended with great relief. The only drawback is the taste of the solution, which with some people is extremely disagreeable; this objection, however, may be overcome by causing the patient to rinse out the mouth with a little eau-de-Cologne and water immediately before and after the application. I also recommend the same kind of iodized solution, mixed, however, with a little chloroform, to be rubbed into the skin on the front part of the neck twice a day; the object is not to produce counter-irritation, but to obtain an absorption of the iodine. Where the soft parts of the larynx are much swollen and hardened, scarifying the part with

Mackenzie's laryngeal scarifier sometimes gives relief; this means is especially useful when the epiglottis is enlarged, producing dysphagia and much pain in the larynx. A few punctures of the epiglottis let out a little (though very little) blood, and give much relief at the time. Care should be taken, however, not to repeat the punctures too often, lest waste of substance and softening of the tissue should occur subsequently, at the punctured spot. I have never observed purulent matter discharged by puncturing the larynx in laryngeal phthisis; the small lancet is felt to enter a hard gritty tissue, and a drop of blood will appear at the puncture; so little blood is usually obtained that it hardly tinges the saliva on clearing the throat; on a subsequent examination with the laryngoscope at the same sitting, a little red speck, but no oozing of blood, is all that is likely to be seen of the puncture.

I have never found leeches applied externally to the throat of much use, and as to counter-irritation, I am inclined to doubt whether any benefit be derived from it in such cases. With respect to the aphonia, I am afraid no other means is available but Faradization of the vocal cords although the voice may return for a few seconds after scarification. Where the laryngeal mucous membrane is but slightly thickened, the aphonia resulting mostly from a state of debility of the cords, electricity may be of marked benefit, and often after a few sittings words and sentences can be spoken out at intervals. It is better to place the electrodes of the battery outside, right and left of the thyroid cartilage (a method of applying electricity to the larynx suggested to me by Dr. Krishaber of Paris), than introduce one of the electrodes into the larynx, as is done in cases of hysterical aphonia. I usually move about the wet sponges on both sides of the larynx so as to avoid exhausting the muscles by prolonged excitation. The operation may be repeated from twenty minutes to half an hour every other day; of course it is only in chronic cases that electricity can be applied; where the larynx is greatly inflamed and very painful, no such treatment is admissible.

Climate.—The climate most likely to benefit cases of laryngeal phthisis is of course that which is best suited to the treatment of phthisis. At a certain height above the sea, a height which varies with the latitude, no cases of phthisis, or but very few indeed, are to be met with. Dr. Lombard, of Geneva ('Climat des Montagnes'), places that height in Switzerland at a little beyond three thousand feet, an altitude also adopted by Dr. Hermann Weber ('Med.-Chir. Trans.,' 1862). Dr. Symes Thompson ('Med.-Chir. Trans.,' 1873,) considers that temperature, as well as altitude and latitude, should be taken into account with respect to a place fit or unfit to be a health resort.

We have yet, I think, to test experimentally how far it is advantageous to send patients to winter in the mountains in northern countries at those altitudes where consumption is unknown, although such places may be admirably adapted for summer residence. We have also to find out to what extent the conditions producing this immunity from phthisis in the native population influence the arrest of phthisis where it has clearly broken out.

I am inclined to believe that, as a rule, those who suffer from consumption, in an acute form, wherever they may be living, except when residing at a considerable elevation, will derive benefit from change to a higher situation, although perhaps not distant from their previous residence, and possibly not more than from 100 to 300 feet above it. Thus an obvious benefit is not unfrequently met with by removing merely from London to Norwood. From Geneva, 1200 feet above the sea, patients are frequently sent in the spring-time to Mornex, 500 feet higher, on Mount Salève, where they often improve in a most remarkable way. On the Mediterranean coast I have seen cases of pneumonia, hæmoptysis, and fever, not only get better, but recover completely in a very short space of time by leaving the seaside and resorting to the hills a short distance from the sea, at a difference of altitude ranging from 150 to 300 feet. I have even met with a patient suffering from acute phthisis, at Nice, who derived temporary benefit from being moved from the first floor to

the top floor of a tolerably high hotel; while, on the other hand, I have known patients to be the worse for coming down to the Mediterranean coast in the spring, after enjoying tolerably fair health throughout the winter on the hills.

In cases of chronic phthisis, with the pulse and temperature normal, it may perhaps be an advantage for patients to take up their residence nearer to the Mediterranean coast on a low level; but should the pulse increase in frequency, and the temperature of the body rise, the hills but a little way off should be resorted to at once.

Syphilitic or malignant disease may be mistaken for laryngeal phthisis. The absence of the physical signs of pulmonary consumption will, as a rule, dispel the idea of a tubercular condition of the larynx; still, as the symptoms of phthisis are not always very obvious, it will perhaps be well to pause for a little time before deciding on the treatment. A course of mercurial medicine advisable in one case would be likely to do mischief in the other; indeed, according to my own experience, all mercurial preparations should be carefully withheld in tubercular disease, whether of the lungs or throat. It is not easy to point out any very marked difference between the laryngoscopic appearance of syphilitic and tubercular laryngitis; these two kinds of affections I have seen, indeed, present like appearances; perhaps the syphilitic disease is more liable to spread to the uvula and velum, to produce thickening and ulceration, and interfere with the respiration at the glottis. The history of the case will of course help to "settle the question." Epithelioma of the larynx will exhibit warty excrescences in the part, easily recognised by their angry-looking appearance, and the intense pain felt in the throat; there may be in such cases much dysphagia and pain on deglutition, without any affection of the epiglottis.

Post-mortem appearance.—Towards the fatal termination of consumption, softening of the mucous membrane of the larynx sets in, where there has been laryngeal phthisis, and after death the mucous membrane of the larynx

discloses the pitted character observed during life. I have before me the notes of the post-mortem appearances of the larynx of a boy aged sixteen years, who died of consumption with laryngeal phthisis at the Brompton Hospital. Softening had commenced on the right arytenoid body, which exhibited an irregular wasting of tissues, and depressions with ragged edges on the mucous membrane. The surface of the larynx appeared to have been undergoing a process of solution and removal; the epiglottis was studded with translucent specks, as if the substance deposited was undergoing a change preparatory to actual softening; the false cords appeared healthy, the true cords exhibited an injected and swollen appearance. In another case, that of a girl of twenty-two years, the whole of the posterior wall of the larynx was found uniformly tinged with a pink colour; softening had commenced under the epiglottis at the commissure between the anterior ends of the vocal cords.

Softening may extend down the trachea, or perhaps begin in the trachea, reaching the larynx; it is, of course, difficult to tell where the affection commences.

Cases.

Before proceeding to offer a few remarks on the cases recorded at the end of the present communication, I shall beg leave to give an account of two patients, which appears to me of interest with respect to the subject of the present paper. One of them exhibited a very suspicious appearance of phthisis in the larynx, while the physical signs of the chest were not those of perfect health. The other suffered from acute laryngitis, with aphonia accompanying congestion at the base of the lungs. Both patients recovered perfectly.

B. H—, æt. 17 years, had been an inmate of my own house for a period of seven years, and was therefore constantly under my observation during that time. He was tall and well proportioned, and his general state of health was excellent. On the 6th April, 1871, he complained to me that he felt a thickening in his throat as if a ball was lodged there. This peculiar sensation he had first noticed on the 3rd of that month; he had also coughed up a little blood on that same day (the 3rd April). On looking into his throat with the laryngoscope, I distinctly saw that his epiglottis was swollen; he complained of pain in swallowing, which (as usual in such cases) he referred to the inside of his ears, and his voice was rather weaker than usual.

Physical signs of the chest.—Left side harsh, bronchial, noisy expiration; at right apex shade of dulness to second rib and vocal resonance deficient over that space, but no crepitation; respiratory murmur normal. At right supra-spinous fossa respiration very harsh; murmur bronchial and deficient; no dulness; a systolic bruit at apex of the heart. Pulse 76, regular; weight of body 8 st. 11¼ lb. Never suffered from rheumatism; was formerly subject to abscesses. When six years old he had, he says, brain fever, and was ten days without speaking a word. Never suffered from inflammation of the lungs, or cold or catarrh of any kind; there is no hereditary predisposition to phthisis. I treated the case with syrup of iodide of iron, cod-liver oil, and his throat was rubbed externally with iodized oil.

On the 12th April, or six days after commencing the treatment, he had gained 2¾ lb. weight.

On the 19th his throat was much better, the peculiar sensation he first complained of having much diminished. Coughed a good deal and expectorated; right apex respiration very harsh and some dulness to second rib confirmed. Left in front nothing abnormal; back of right side respiration very harsh, and dulness at apex. Applied acupuncture and a weak solution of croton oil in cajuput oil to right apex.

Only a slight degree of thickening of the epiglottis now remained, and he felt but little uneasiness in the throat on swallowing.

27th.—No longer any discomfort in the throat excepting in a slight degree in the evening when going to bed.

Physical signs.—Questionable dulness right apex; murmur rather bronchial at that spot with somewhat exaggerated expiration; no crepitus; he had lost, however, 1¼ lb. weight.

May 15th.—Right apex quite clear on percussion, but a shade of dulness at the back where murmur is deficient and very harsh. Now feels quite well, and can take a long walk without fatigue.

November, 1871, at Nice.—Respiration rather louder than natural in the right side; murmur deficient and harsh with distinct crepitus on the right side at the back; a decided though but slight dulness at right supra-spinous fossa. Had been in very good health since last May; epiglottis was seen to have resumed its normal state, though slightly thickened at the rim. No cardiac murmur whatever, but cardiac impulse rather strong.

December 6th, 1872, Nice.—Had spent last winter at Nice, and the following summer in England. Percussion note on both sides now quite resonant; left and right supra-spinous fossæ respiration bronchial, very harsh, with loud expiration. Complained of sore-throat; had had a chill three or four days before. Pharynx red with some small aphthæ on the pillars. The epiglottis at the tip appeared a little thicker than usual. Weight 9 st. 9 lb., had gained 9¾ lb. since the 19th April, 1871.

March 13th, 1874.—Had again wintered at Nice; no abnormal signs in front left and right. At the back very fine crepitation right supra-spinous fossa, and respiration harsh lower down. Left supra-spinous fossa respiration harsh, normal lower down: no dulness. A laryngoscopic examination showed the epiglottis to be still slightly thickened at the rim, but free from congestion or irritation. Said he now felt nothing the matter with his throat; indeed, had

enjoyed excellent health since last examination. He had grown into manhood, aud his frame was well developed in every respect. Now weighed 10 st. 5¼ lb., having increased 10½ lb. since December, 1872.

This case is interesting, and certainly looks very much like one of phthisis arrested *ab ovo*. The epiglottis is not, according to my experience, liable to swell from common temporary inflammation, as would be the case with the other parts of the larynx in laryngitis. An enlarged and indurated epiglottis is to me an invariable sign of some serious mischief. In the present instance there may still remain a very slight thickening of the epiglottis. I have no record of ever observing the white mucous secretion in this patient's larynx, or, indeed, of any other laryngeal mischief besides that relating to the epiglottis.

In the following case there were pulmonary congestion and bronchial catarrh in the lower part of the lungs, and in the larynx the vocal cords were mainly affected, the epiglottis remaining healthy.

Mr. B—, aged about 40, arrived at Nice suffering from incessant and very loud coughing, and having entirely lost his voice. He caught cold about eight weeks before from having been detained at sea in a small boat after dark, when he had to row for a considerable length of time, and was probably chilled after perspiring very freely, and when tired in other respects. His throat became sore, and he was treated with the application of nitrate of silver, but his voice became weaker and dropped into a whisper.

January 16th, 1874.—His cough comes in violent noisy fits; there is some little expectoration at night, but none in the daytime.

19th.—Pulse 102; temperature under tongue 101°; much wheezing heard under the sternum. Face red; cough distressing; larynx had been found on a previous occasion red and angry, though not apparently swollen. I could not distinguish the true vocal cords, and as medical treatment

appeared of little use, weak as he was and but just able to stand, I took this patient out of his bed up to a villa at St. Philippe, about 150 feet above Nice, on the slope at the western side of the town. The next day he was already much better, his voice returning at intervals, though weak and hoarse. Appetite returning. Temperature of the body, 99·4°; pulse, 100.

29th.—Improvement continued. Had walked an hour without fatigue; appetite much better. I then obtained a good view of the throat. On attempting to make a sound there was some difficulty in the adduction of the cords, which sometimes met, sometimes remained apart. During phonation I could plainly see the true cords reduced to a very thin pale lamina, instead of exhibiting their white pearl glistening bands. The false cords appeared rather thickened; voice still very hoarse and rough. On January 19th the wheezing under the sternum had entirely disappeared, but there remained some distinct crepitation with slight dulness over a considerable portion of the base of the left lung.

On January 29th dulness had disappeared, though crepitus continued.

February 3rd.—Rudimentary vocal cords approached each other, but lacked proper tension; they appeared shortened and did not vibrate freely in the act of phonation.

16th.—Vocal cords returning to their normal state, voice much improved; murmur more natural at left base and less crepitus.

25th.—Vocal cords had almost perfectly recovered.

March 4th.—Could take long walks of three or four hours without fatigue; voice normal. Left the villa on the 11th of March.

The following are the weights of this patient as taken with a reliable weighing chair:

January 31.—12	stone	2½	pounds.
February 3.—12	„	3¼	„
„ 9.—12	„	5¼	„
„ 12.—12	„	6¼	„

February 16.—12	„	7¾	pounds.
„ 19.—12	„	7½	„
„ 25.—12	„	7	„
March 4.—12	„	9½	„

A gain of seven pounds weight from the 31st January to the 4th March.

The beneficial effect of the pure bracing air of the hills, although only about 150 feet above the sea, was most remarkable in the present case, the improvement in the state of the larynx progressing together with that of the chest.

I have reported below in an abstract form seventy hospital cases, most of them occurring amongst my own out-patients at the Brompton Hospital, a few others having been sent to me by my colleagues. These cases were followed up as far as possible, but it is difficult to induce hospital out-patients to attend for a lengthened period; so that many were lost sight of at a time when I was most anxious to ascertain their state of health.

Remarks on the Cases.

The number of male patients far exceeds that of the female patients; there being 49 of the former, and 21 of the latter. I do not recollect exercising any selection in my cases of laryngeal phthisis, and my impression is that I noted down all cases of phthisis with laryngeal complication, while engaged with the present inquiry. I therefore believe there is a fair ground for concluding that men are more liable to laryngeal phthisis than woman.

The ages were recorded in 55 cases. They are as follows:

Number of cases aged	20	and	under	.	.	4
„	between 20	„	30	.	.	26
„	„ 30	„	40	.	.	16
„	above 40	.	.	.	.	9

This seems to show that age has little or no influence as a

predisposing cause of laryngeal phthisis, as, according to Dr. J. E. Pollock,[1] there is a large preponderance of cases of phthisis between the ages of 20 and 30. Therefore, as far as age is concerned, the predisposition appears to apply equally to consumption, whether the throat be affected or not.

Out of the 70 cases, 8 exhibit no visible organic change in the larynx (slight congestion and redness in 2); these cannot be considered strictly as cases of laryngeal phthisis, but should be regarded as cases of functional defects of the larynx in phthisis. In 1 of these 8 cases the organ was apparently affected long before consumption set in; therefore, 7 only can be really considered as connected with tubercular disease of the lungs. The 8 cases are as follows:

Case No. 1.—Cavern.	Adduction of cords deficient.
„ 13.—Dulness, metallic crackling .	Ditto.
„ 21.—Crep., dulness, and crackling .	Tension of cords deficient.
„ 30.—Cav. respiration and crackling.	Adduction of cords deficient (some redness of larynx, but cords normal).
„ 35.—Harsh respiration and prolonged expiration, shade of dulness; hæmoptysis and strong hereditary predisposition . .	Tension of cords deficient.
„ 36.—Cav. and crackling, dulness .	Tension deficient, adduction normal.
„ 46.—Falling in at right apex, dulness, crepitus, hæmoptysis .	Tension deficient.
„ 55.—Metallic click, coarse crepitus .	Ditto.

Tension of the vocal cords was therefore deficient in five cases, and adduction of the cords was deficient in three.

As to the actual change which had taken place in the larynx, in 31 of the 70 cases, the epiglottis was observed to be affected.

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[1] 'Second Medical Report of the Hospital for Consumption,' 1863; and 'Treatise on Prognosis in Consumption.'

The following are the alterations this body was found to exhibit:

Case No.	2.	Epiglottis,	knotted and atrophied.
„	3.	„	concave at margin.
„	6.	„	thickened and studded with granulations.
„	8.	„	pendulous, misshapen from induration and softening.
„	11.	„	much swollen and pendulous.
„	12.	„	very red and subsequently granular, though not swollen.
„	16.	„	streaked from congested capillaries, but neither swollen nor granular.
„	22.	„	much swollen and curled, and exhibits tubercular granulations.
„	23.	„	small, speckled and appeared white (probably pale).
„	24.	„	swollen and pendulous.
„	28.	„	red, pendulous, and contracted into a cylindrical shape.
„	29.	„	pendulous, notched at apex, much swollen and speckled.
„	32.	„	pale, thickened, and lobulated.
„	33.	„	red, but not swollen.
„	34.	„	thickened and granular, becoming subsequently much swelled.
„	40.	„	swollen.
„	41.	„	indistinctly granular.
„	42.	„	much swollen and red, becoming pale, contracted, and indurated after scarification.
„	44.	„	red, streaked, and granular.
„	47.	„	pendulous and streaked.
„	48.	„	large and pendulous.
„	49.	„	red, granular, shortened, much swollen.

Case No.	50.	Epiglottis,	very red, swollen.
,,	51.	,,	pale and thickened.
,,	53.	,,	pendulous, swollen and tubercular.
,,	54.	,,	red, but not swollen. (Syphilitic taint.)
,,	56.	,,	pendulous, red, and much thickened.
,,	57.	,,	pendulous, swollen, and granular.
,,	61.	,,	pendulous and tubercular.
,,	64.	,,	very red, and congested.
,,	69.	,,	pendulous, much swollen, and tubercular.

I find only thirteen cases (about 18·5 per cent.) of positive ulceration of the larynx, which appears to be a small proportion. Patients thus affected are, as a rule, older than those whose larynx are not ulcerated; the mean age of the cases of laryngeal disease without ulceration being 29·7 years, while that of the cases of laryngeal disease with ulceration is thirty-eight years. I think it is, therefore, fair to conclude that young consumptive patients are less liable to laryngeal ulceration than their seniors; and that this pathological change is superadded in a swollen and tubercular larynx, to some extent on account of the diminished vitality and want of power of nutrition depending upon age.

The other prominent organic change is, as a rule, a state of swelling of the arytenoid bodies and epiglottidean folds; the vocal cords being often obscured by the hypertrophied false cords, and reduced by congestion to a narrow rim. The white secretion I have referred to is frequently witnessed covering the swollen and tubercular mucous membrane.

As to the dysphagia and the alterations in the voice, and the cough: I have noted difficulty and pain in swallowing in fourteen cases, but this symptom may not have been recorded in others. It is caused, as a rule, by a swollen and tubercular condition of the epiglottis; but deglutition is sometimes interfered with, although the epiglottis remains in the healthy state. During the act of swallowing the larynx is

raised, the root of the tongue resting like a cushion on the epiglottis, which covers the laryngeal opening; thus the tongue, together with the constrictor muscles of the pharynx, presses upon the swollen epiglottis and larynx, and elicits in some cases intense pain. Loss of voice varied from mere weakness of the voice to hoarseness and complete aphonia. In some few cases the aphonia had an intermittent character; in most of them it was either complete or nearly so; but the patients could always speak in a whisper. Of the 70 cases recorded in the table, 59 are stated as exhibiting some alteration of the voice. In by far the greater number it was extremely feeble, in a few it was merely weak, and with a few others it was hoarse or cracked. In the other 11 cases, although the condition of the voice was not recorded, it may be concluded from the appearance of the larynx that the voice was probably either weak or otherwise affected in nine, and there was apparently nothing wrong with the act of phonation in two cases only.

SEVENTY CASES OF LARYNGEAL DISEASE IN PHTHISIS.

(M. indicates male; F. female.)

1.—M., æt. 25.

State of the—

Lungs.—Left apex, cavernous respiration, with loud friction sound and slight dulness; very little natural murmur. Right side, respiration harsh, with crackling.

Larynx.—Weakness of adductor muscles of cords only. Ill for 18 months; aphonia for the last 2 months.

2.—F., æt. 25.

Lungs.—Left, noisy respiration and moist râles, but little natural murmur. Right, cavernous, blowing, and dull at apex.

Larynx.—Epiglottis knotted and atrophied; larynx swollen; impeded movements of glottis.

3.—F.

Lungs.—Right, apex at back, a cavity, left, crackling at the side.

Larynx.—Epiglottis concave at margin; impeded movement of glottis; vocal cords red and swollen.

4.—M.

Lungs.—Cavernous.

Larynx.—Laryngeal mucous membrane wrinkled and relaxed; hoarseness for two months.

5.—M., æt. 30.

Lungs.—Left, respiration very feeble. Right, respiration weak; fine mucous râles. Posterior left, vesicular murmur right supra-spinal fossa; respiration deficient and very harsh, dulness. Hæmoptysis in streaks.

Larynx.—Raised papillæ base of tongue; arytenoid bodies and arytenoidian folds ulcerated; movements of glottis free, but vocal cords pale and lustreless; aphonia, or very hoarse for 8 or 10 months.

6.—M., æt. 43.

Lungs cavernous, with softening and physical signs of chronic second and third stage of phthisis.

Larynx.—Enlarged follicles base of tongue; epiglottis thickened and granular; larynx swollen; aphonia; temporary recovery of voice from treatment.

7.—M.

Lungs.—Left, apex dull, with diffused crepitus in front. Right, some crepitus, with deficient respiration.

Larynx.—Left side, deep ulceration, extending to posterior end of vocal cord; epiglottidean folds red and much swollen; apparent loss of power of tension; epiglottis pendulous; voice affected for 14 months, viz. hoarseness for 10 months, then aphonia.

8.—M., æt. about 26.

Lungs, March 15th, 1872 (Nice).—Left front, humid crepitus; murmur very deficient; shade of dulness at clavicle; posterior left, humid crepitus; some dulness throughout. Right, respiration fair.

Larynx, March 15th, 1872.—Epiglottis pendulous, out of shape from induration and softening; no possible view of larynx; voice returned to some extent under treatment; aphonia for about 6 months. Died March, 1873.

9.—M., æt. 40.

Lungs.—Left quite dull on percussion; crepitus. Right, shade of dulness; no hæmoptysis.

Larynx.—No cords visible; larynx thickened, pain on deglutition, and at one time serious dysphagia; aphonia for 22 months; pendulous epiglottis; suffered much from the throat for the latter 4 months.

10.—M., æt. 26.

Lungs.—Softening; right side dull, with tubular breathing. Left, tubular respiration; respiration very harsh, no dulness.

Larynx.—Rudiments of vocal cords only, the right presented an ulcerated-looking margin; larynx swollen, and the whole cavity contracted on attempts at phonation. Began losing voice about 10 months ago; aphonia for last 7 months.

11.—M.

Lungs.—Right side, some crepitus with bronchial breathing, and a shade of dulness near the sternum; crackling. Left, healthy. No syphilitic taint.

Larynx.—Epiglottis much swollen and pendulous, concealing larynx. Sudden aphonia about three months ago after exposure to a draft; for a fortnight in excruciating pain on swallowing alcoholic fluids or beer; with an effort some words were spoken in a tolerably natural voice.

12.—F., middle-aged.

Lungs, Oct. 31st, 1868.—Right, crackling at apex, with slight dulness. May 3rd, 1869.—Left apex, a large dry cavity; and right, dulness with cracking and very little respiration.

Larynx, Oct. 3rd.—Epiglottis very red; arytenoid bodies, folds and false cords red and swollen; secretion of white mucus in larynx; vocal cords much congested and their lustre lost. Singing power failed 16 months before; voice tolerably clear at this time. May 3rd, 1869.—A speckled appearance of

the larynx. May 22nd.—Epiglottis appeared granular, not swollen; movements of cord free; no swelling beyond slight apparent thickening.

13.—M., æt. 22.

Lungs.—Left and right, shade of dulness; left, supra-spinal fossa, metallic crackling. First signs of phthisis set in with pleurisy about 13 months ago.

Larynx.—Case of deficient tension and some congestion of vocal cords, with red and relaxed mucous membrane.

14.—M., æt. 44.

Lungs, end of Sept., 1868.—Right, shade of dulness; no obvious signs of phthisis. Nov. 9th, 1868.—Right, apparently distant amphoric respiration; right, back, decided dulness. Jan. 18, 1869.—Left, in front, respiration deficient and very harsh. Right, murmur feeble; at the back right, dulness thoughout.

Larynx, end of Sept., 1868.—Laryngeal space much reduced from thickening; mere rudiments of vocal cords left, at posterior ends of which were deep ulcerations. January 18th, 1869.—Right epiglottidean folds and arytenoid bodies much swollen; some relief from scarification. Complete aphonia for last three years. (Had partial aphonia 8 or 9 years ago; relieved occasionally, especially in the summer, till 3 years ago.) Pain in larynx till February, 1869, when deglutition became less free than before.

15.—M., æt. 35.

Lungs.—A large dry cavern, extending from apex to third rib on right side (dulness and cavernous respiration).

Larynx.—Back of tongue red and injected; follicles enlarged; epiglottis normal; arytenoid bodies and folds swollen; false cords swollen and true cords less glistening than usual; loss of tension of cords, and speech carried on in a low, harsh tone.

16.—M., æt. 37.

Lungs, 30th Oct.—Mucous râles; no dulness; no cavernous or bronchial breathing. Nov. 7th.—Shade of dulness, left apex. 28th Nov.—Slight but distinct dulness, left apex, in front. 13th Feb., 1869.—Left apex, respiration tubular with slight crepitation and dulness, and amphoric breathing at right supra-spinous fossa.

Larynx, 30th Oct.—Thick and white mucous secretion over epiglottis and folds, visible also in the larynx; no congestion or granular appearance; no pain, no hoarseness, but a constant desire to clear the throat. 13th Feb.—Epiglottis streaked deeply from congested capillaries; walls of larynx red, but no longer any white secretion to be seen; voice occasionally weak, no aphonia.

17.—M., middle-aged.

Lungs, 3rd Nov., 1868.—Bronchial breathing; crackling at apex; right fine crepitus and mucous râles extensive; no dulness.

Larynx, 3rd Nov.—Back of tongue red, with thick white secretion upon it;

larynx particularly pale and secreting thick white substance; vocal cords healthy.

18.—M., æt. 38.

Lungs.—Left, in front, respiration harsh, with thick mucous râles; no other signs; questionable signs of phthisis.

Larynx swollen and vocal cords not seen; on attempt at phonation the folds of mucous membrane project towards median line; white secretion on the posterior wall of larynx, the removal of which brings into view a pale and relaxed mucous membrane. Sudden aphonia 6 weeks ago, and no return of the voice.

19.—F., æt. 22.

Lungs, Oct., 1868.—Amphoric respiration right apex, with crackling and dulness (cav.). 14th Dec.—Symptoms urgent; pulse 144; temperature under tongue 103·2° F.

Larynx, 9th Oct., 1868.—On attempting phonation the cords did not meet well in median line; ulcerated raised patch on posterior wall, studded with enlarged follicles. Aphonia for 3 months; sometimes voice broke out hoarsely for a minute or two. 16th Nov.—Mischief in larynx increasing; ulcerations distinctly seen.

20.—M., æt. 51.

Lungs, 18th Nov.—Shade of dulness, left apex. 7th Dec.—Decided dulness; respiration harsh, left and right. 17th Dec.—Left apex dull to third rib; distant metallic sounds. Severe hæmoptysis 4 years ago.

Larynx.—A good view of larynx with assistance of ice bag to throat and ice in mouth; larynx swollen near posterior end of left cord, a raised ulcerated patch stretching across posterior wall of larynx but not reaching right cord; enlarged follicles on the raised membrane. Had a cracked, reedy voice; words often dropped; movement of the cords appeared free.

21.—F., æt. about 28.

Lungs, 19th Nov., 1869.—At upper third left side, in front, deficient murmur; abundant mucous râles. Dec. 9th.—Left, crepitus; right, crepitus and dulness under clavicle. Dec. 23rd.—Left side, crackling. Jan. 22nd.—Physical signs of softening still were obvious.

Larynx, Dec. 2nd.—Voice became natural after an application of a solution of nitrate of silver to larynx. Dec. 11th.—Epiglottis and larynx red and swollen; voice natural. Had aphonia 3½ years previously for about 10 weeks. A severe attack of hæmoptysis 4 years ago.

22.—M.

Lungs.—Left, respiration tubular; right, mucous râles and deficient respiration; murmur; no dulness; hæmoptysis and expectoration considerable.

Larynx.—Epiglottis much swollen and curled; exhibits tubercular granulations; arytenoid body much enlarged, ragged, and speckled; laryngeal space considerably narrowed; such pain that he could hardly swallow; aphonia. Voice

returned a few days after application to me, and deglutition improved, but thinner and weaker in other respects.

23.—F., æt. 27.

Lungs, first examination, about 20th November, 1868.—Shade of dulness, left apex. Dec. 2nd.—Harsh breathing (physical signs not clearly those of phthisis, but the state of throat left little doubt).

Larynx, about 21st Nov.—Has a high-pitch cracked voice; cords approach, though not freely, on phonation; epiglottis small, speckled, looks white (pale); some white secretion on false cords; want of power in adductor muscles of cords (state of larynx symptomatic of phthisis).

24.—M., æt. 38.

Lungs.—Left, in front, consolidation and softening; posterior left, cavern; right, also dulness and cavernous respiration. Father, mother, and brothers died of phthisis. Began coughing last Christmas; in good health before.

Larynx.—With use of ice-bag. Epiglottis observed swollen and pendulous; arytenoid bodies and folds considerably swollen, and secretion of white substance issuing between arytenoids; no visible ulceration; swallowing occasionally produces spasm of the glottis. Voice first failed 3 months ago. At present aphonia, though voice returns slightly at times.

25.—M., æt. 25.

Lungs, 19th Dec., 1868.—Left side; some dulness. 12th Jan., 1869.—Left, loud dry cavernous breathing, with occasional click.

Larynx, Dec. 11th, 1868.—Both sides of larynx very much swollen and covered with white mucus; epiglottis not thickened; swelling punctured. Dec. 16th.—Less swelling, but white mucus continues present; an indistinct ulceration left. 21st Dec.—Epiglottis not swollen, but contracting in some respects; much white mucus. Jan. 25th.—A conical tumour or growth on right arytenoid body. Supraglottic region softening. Loss of voice about 11 months before applying. Had had no food for 4 days when I saw him, from inability to swallow, and was starving; but drank some milk and sherry after scarification of the larynx. Died Jan. 25th, 1869.

26.—F.

Lungs.—(Cavern and softening). Left, some coarse crackling; right, amphoric breathing and dulness at apex to third rib; falling-in of chest at right apex; a chronic case.

Larynx.—Sound of voice imperfect; arytenoid bodies fail to approximate sufficiently in the act of phonation; a raised patch between arytenoids is covered with white secretion, on removal of which follicles like spiculæ are seen.

27.—M., æt. 24.

Lungs.—Consolidation and softening; a large tumour, feeling tense and elastic, but without fluctuation, just under left false rib, measuring in the

erect posture, 4½ inches horizontally and 2¾ inches in breadth. This resulted from his being squeezed between a waggon and dust-cart in Sept., 1866.

Larynx, 4th Jan., 1869.—Arytenoid bodies and folds much swollen and secreting white mucus; epiglottis not enlarged; an ulceration on left false cord extending to true cord on that side; aphonia for nearly a year. 16th Jan.—Slight return of voice; ulceration appeared to have a tendency to cicatrize; felt better.

28.—M., middle-aged.

Lungs, 16th Jan., 1869.—Left, respiration deficient and harsh; right, shade of dulness at apex. Phthisis probable.

Larynx, 16th Nov., 1868.—Epiglottis red, pendulous, and contracted into a cylindrical shape; mucous membrane not readily visible on account of epiglottis, but appeared thickened. Lost voice on 8th July last, when he coughed up about half a pint of blood. Voice began improving about end of October following. Stated that since 8th July he had gained 11 lbs. weight.

29.—M., æt. 22,

Lungs, 23rd Jan., 1869.—Left, dry cavernous respiration from apex to third rib, with occasional crackling; from third rib to base distant cavernous breathing. Right, amphoric sounds front and back. Severe attack of inflammation of the lungs about 6 years ago.

Larynx, 23rd Jan., 1869.—Back of tongue very red and follicular; epiglottis pendulous, notched at apex, much swollen, and speckled; exhibited white secretion, which was also seen oozing out of larynx; great pain on deglutition. Jan. 27th.—After scarifying the epiglottis obtained glimpse of larynx, which exuded a white substance in abundance. Aphonia since the end of last August; began with hoarseness for 2 or 3 months. Two years ago, hæmoptysis; was first troubled in singing, but was able to sing till May last.

30.—F., æt. 30.

Lungs, 5th Feb., 1869.—Right apex, in front and behind, cavernous respiration and crackling.

Larynx, 5th Feb., 1869.—Larynx red, with vocal cords healthy; want of power in adduction; weak, nervous, subject to fainting. Voice lost 4 months ago; it returned after Faradization, and continued fair, though rather weak, till March 4th.

31.—F., æt. 23.

Lungs.—Left, base, shade of dulness; right, cavity, frequent hæmoptysis. Mother died of phthisis.

Larynx.—Epiglottis normal; on posterior wall of larynx a cluster of raised follicles. Aphonia for last 15 or 16 months; began coughing about 3 years ago; probably adduction deficient.

32.—F., æt. 22.

Lungs, 17th Feb., 1869.—Abundant crepitus left and right, murmu

chial. (Taking into account laryngeal symptoms, softening probably in progress.)

Larynx, 17th Feb., 1869.—Root of palate pale, and back of pharynx exhibited small superficial excoriations; uvula out of shape and speckled with granulations; epiglottis pale, thickened, and lobulated; much dysphagia; no relief from scarification; epiglottidean folds appeared swollen.

33.—M., æt. 32.

Lungs, 20th Feb., 1869.—Left, tubular respiration and moist crepitus at base. Had suffered from hæmoptysis.

Larynx, 20th Feb., 1869.—Ulceration at base of both arytenoid bodies externally to larynx, so that they look like pedicellated tumours, are granular and very red; epiglottis red, but not swollen; no pain on deglutition. Sudden aphonia on 8th January last; coughs since last November. Treatment—Liquor Arsenicalis; cod liver oil and iodized oil to larynx. 25th Feb.—Throat felt much better; voice returned, though very hoarse.

34.—M., æt. 43.

Lungs.—Began, with uncertain signs of phthisis, on 27th June, 1868. Oct. 2nd, 1868.—No positive signs of phthisis yet. Nov. 4th, 1868.—Metallic crackling, first sign, observed.

Larynx, 27th June, 1868.—Follicles back of tongue red and enlarged; epiglottis thickened and granular; mucous membrane of larynx much swollen. Loss of voice in October, 1867; began with sore-throat, which lasted for 3 months before aphonia became complete. (Never recovered perfectly from pleurisy in 1866; slight hæmoptysis once or twice in March, 1868.) Oct. 16th, 1868.—Epiglottis much reduced in size, but still speckled with granulation. Died 10th Nov., 1869. (See 'Clinical Notes on Diseases of the Larynx,' p. 99.)

35.—F.

Lungs, 5th March, 1869.—Harsh respiration and percussion note not very clear. Two slight attacks of hæmoptysis last 6 weeks. Mother, three sisters, and two brothers died of consumption. 26th March.—Left, harsh respiration; no dulness. Right, harsh breathing, prolonged expiration, and shade of dulness at apex.

Larynx, 14th Jan., 1869.—Cords look quite healthy, and adduction quite free, but loss of tension; the whole larynx contracted in efforts to produce sounds. April 5th, 1869.—Has improved gradually; voice now natural, and can even sing a little. Treated by Faradization.

36.—M. 21.

Lungs.—Right apex, in front, a shade of dulness; cavernous respiration with crackling to third rib; base, respiration vesicular, with some crepitus; back, right side, unmistakable signs of cavern and consolidisation ('Clinical Notes on Diseases of the Larynx,' p. 134).

Larynx, 19th March.—Defective tension and abnormal adduction, producing cracked voice. Recovery by Faradization, and could sing the scale without difficulty on the 8th July following.

37.—F., æt. 27.

Lungs.—Cavernous respiration and prolonged expiration, left side, between third and fourth rib; right, falling in (slight) at apex and crackling.

Larynx, 1st May, 1869.—No laryngeal swelling; no swollen follicles; no ulceration; supraglottic mucous membrane red and relaxed, especially right side; none or mere remnants of vocal cords visible; posterior wall of pharynx coated with white secretion; phonation required great effort, and apparently carried on with false cords. 1st Nov.—Left, arytenoid body red and swollen; pain in inside of left ear; epiglottis rather pendulous, but appeared healthy. Only temporary benefit from Faradization.

38.—M., æt. 36.

Lungs, 6th April, 1869.—Left, crepitus; prolonged respiration. Right, dulness and tubular respiration. Hæmoptysis, 18 months ago.

Larynx, April 6th, 1869.—Epiglottis partly pendulous; larynx congested; congestion and loss of power in vocal cords. May, 1869.—Left cord appeared uneven and atrophied; a chink between cords on attempting phonation. Aphonia for 6 months, either total or voice very hoarse. Affection very sudden; patient went to bed quite well, and was voiceless next morning.

39.—F., æt. 37.

Lungs, 18th May, 1869. — Left, fine crepitus; shade of dulness under clavicle. Right apex, breathing rather bronchial. Slight hæmoptysis once or twice. Mother died of phthisis.

Larynx, 18th May, 1869.—Had a string of ulcerations on right epiglottidean fold, which was swollen; adduction too imperfect to allow of phonation. Sept. 21st.—Voice returned at times. Oct. 5th.—Health much improved and voice much steadier. Loss of voice from about 16th April. Treatment with inhalation of iodine.

40.—M., æt. 19.

Lungs, 26th Jan., 1869. — Right apex, tubular breathing; posteriorly right, dulness; left, normal. Hæmoptysis, Dec., 1868; lost 11 lbs. weight in less than 5 months.

Larynx, 14th May, 1869.—Supraglottic region very red, and space contracted from swollen arytenoid bodies and folds. 31st May.—Epiglottis swollen. June 5th.—Adduction incomplete; voice fails now and then in speaking. June 25th—Voice now normal.

41.—M.

Lungs.—Left, in front, distant cavernous sounds; dull posteriorly with cavernous breathing and crackling; no hæmoptysis: no hereditary taint.

Larynx.—Base of tongue follicular; no ulceration; redness and slight

laryngeal thickening; epiglottis indistinctly granular; right arytenoid overlapped left in phonation; cords part when required to vibrate, and voice weak and hoarse in consequence (want of power of tension); no syphilitic taint (an excellent view with the oxyhydrogen light).

42.—M., æt. 27.

Lungs, 29th May.—Left, dull and falling-in, with loud dry amphoric respiration from apex to nipple. Right, dry cavernous respiration.

Larynx, 29th May.—Epiglottis much swollen and red; voice unaffected, indicating normal vocal cords. 2nd July.—Epiglottis secreting white mucus. June 4th.—Epiglottis had become pale, with a tendency to contract. Had been scarified freely on several occasions. June 7th.—Epiglottis smaller, becoming harder and granular. 23rd June.—Epiglottis so far contracted as to allow sight of larynx; cords appeared normal; much white secretion. June 26th.—Voice weak; dysphagia; never any aphonia. Died 11th July, 1869.

43.—M., æt. 37.

Lungs, 29th May, 1869.—Left apex, dulness; slight falling in, absence of murmur and crackling. Right, very deficient respiratory murmur.

Larynx, 15th May.—Right supraglottic region very much swollen; left normal; edges of cord appeared ragged. Complete aphonia since Christmas last. 29th May.—A conical growth seen on right epiglottidean fold; whole larynx very red; no swollen epiglottis; no dysphagia. 2nd June.—Much relief from puncturing right false cord; there appeared a breach of continuity at the seat of a former puncture, as if softening was setting in. June 16th.—Softening appeared commencing on the left of larynx. 25th June.—Left cord much swollen.

44.—M., æt. 31.

Lungs, 29th May, 1869.—Left, crackling; shade of dulness at apex; dulness also right. Hæmoptysis 2 months ago; in ill-health for 12 months. 11th Oct.—A cavity.

Larynx.—Epiglottis red, streaked, granular; larynx very red; arytenoid bodies and folds much swollen; white exudation between arytenoid bodies; voice rather weak, but otherwise natural; occasionally slight pain in swallowing the saliva.

45.—F., æt. 29.

Lungs.—Right apex, in front friction sounds, deficient and harsh respiration. Posterior left harsh, with humid crepitation; some dulness right.

Larynx, 4th June, 1869.—Larynx granular and rather pale; secreting white thick mucus. June 12th.—On attempting phonation, posterior part of larynx drawn up, concealing the vocal cords, but arytenoid bodies move freely towards each other (weakness of tension). June 14th.—Remarkable absence of sensibility of larynx to a strong electrical current. June 30th.—Loud whisper

occasionally after Faradization. There had been total aphonia for 4 months. Deglutition difficult at first, but improved under treatment.

46.—F., middle-aged.

Lungs.—Falling in, with some dulness at right apex; humid crepitation, both left and right side. Hæmoptysis 2 months before.

Larynx.—Case of deficient tension of the vocal cords, without organic disease of the larynx (see p. 13).

47.—M.

Lungs.—Physical signs not reported, but case entered as tubercular; pulse 104; temperature 101·2° F. under tongue.

Larynx, 7th July.—Epiglottis pendulous and streaked, but larynx could be seen red, though not exactly thickened, and exhibiting no white secretion. July 12th.—Vocal cords red and appeared atrophied, approximated tolerably well and vibrated to some extent. For about 18 months either very hoarse or only spoke in a whisper.

48.—M., æt. 32.

Lungs.—Loud tubular breathing and amphoric resonance both apices (Cavity in both apices).

Larynx, 24th Sept.—Larynx red; large pendulous epiglottis, concealing glottis, but a round elevation is visible on left arytenoid body. Very hoarse for last 4 months. 1st Oct.—Laryngeal swelling increased, and covered with milky secretion; it receded before scarifier, and bled only a little; acute pain in the left ear; vocal cords seen red and congested. Said he felt something break in throat two days ago, and expectorated much blood and matter. 25th Nov.—Stated by letter that he was worse and apparently sinking. Treatment by scarification and iodized oil.

49.—F., æt. 24.

Lungs.—Left, respiration apparently tubular; right, dull, flattened, and the seat of cavernous breathing.

Larynx, 6th Oct., 1869.—Epiglottis red, shortened, and much swollen, granular; arytenoid bodies and false cords much swollen; left false cord apparently pitted from softening; much dysphagia; could only swallow a little milk; almost starved. Aphonia complete for 6 weeks. 13th Oct., 1869.—Softening progressing in supraglottic region; epiglottis shortening; rather less dysphagia.

50.—M., æt. 39.

Lungs, 1st June, 1869.—Left, deficient murmur and crackling. Sept. 21st.—Left, in front, bronchial, deficient, and harsh murmur; right, distant, dry, cavernous respiration. Oct. 5th.—Left moist, crepitation throughout, no dulness; right, dull at clavicle. Fell ill about Oct., 1868; an attack of hæmoptysis June, 1869.

Larynx, 21st Sept, 1869.—Epiglottis, vocal cords and supraglottic region

very red and swollen; softening appeared in progress; much white mucus secretion; pain on deglutition referred to inside of ears; throat affection had lasted three months, and was worse now. 5th Oct.—Both arytenoid bodies much swollen, and covered with white secretion. 13th Oct.—Voice weak and falling into a whisper. 23rd Nov.—Could only swallow with great pain. Relief from inhalation of iodine.

51.—M., æt. 49.

Lungs.—Cavities in both lungs.

Larynx.—Both arytenoid bodies, especially the left, very much swollen, exhibited white granulations; epiglottis pale and thickened; an ulceration on posterior wall of larynx, and thick white mucus present. Aphonia for 5 or 6 months.

52.—M., æt. 18.

Lungs.—Cavity in both lungs.

Post-mortem examination.—Pharynx somewhat congested; vocal cords softened; laryngeal mucous membrane terminating abruptly with a plug of mucus at rima glottidis.

Larynx.—Complete aphonia for 4 months; on admission into the Brompton Hospital the larynx was seen to be retracted, white mucus present, and paralysis of tensor muscles of cords. (Notes favoured by Mr. N. R. Smith, Clinical Assistant, Brompton Hospital.)

53.—M., æt. 56.

Lungs.—Loud, dry, cavernous breathing right side; left, respiration loud and vesicular. In fair general health.

Larynx, 18th Nov.—Epiglottis swollen, pendulous and tubercular, though not softening. 24th Nov.—Epiglottis larger, exhibited an ulceration; laryngeal mucous membrane appeared thickened and very red; no vocal cords visible. 29th Nov.—Epiglottis much less swollen, and ulceration cicatrising. A small though rather deep ulceration on the posterior wall of larynx. Was suddenly seized with sore-throat after a chill 4 months ago; at present, aphonia; voice occasionally became very hoarse. Pain in larynx only felt on swallowing or coughing.

Treatment.—Scarification and inhalation of iodine; iodide of potassium and syrup of iodide of iron internally.

54.—M.

Lungs.—Both sides, humid crepitus; right, tubular respiration and falling in of chest. A syphilitic sore about four years before, but no secondary symptoms, except night headache.

Larynx (with syphilitic taint).—Pharynx and back of tongue very red. Uvula nearly white from apparent infiltration with tubercular matter; softening at base, and nearly ulcerated through a quarter of its breadth. Infiltration extended to velum; epiglottis red, but not swollen; right vocal cord

healthy; an ulceration at posterior end of the left cord. Pain followed deglutition. Aphonia for the last five weeks.

55.—M., æt. 22.

Lungs.—Left, metallic click; right, coarse crepitus.

Larynx.—A cracked voice so long as he can remember, viz. when about æt. 3, after measles. Arytenoid bodies rather overlapped each other in phonation, but tension deficient. Adductors instinctively compensated for weakness of tension. (This may have been unconnected with phthisis, but is sufficiently interesting to be placed on record.)

56.—M., æt. 22.

Lungs.—Phthisis, caverns (no notes of physical signs).

Larynx.—Epiglottis pendulous, red, and highly thickened; arytenoid bodies and folds just visible, covered with white mucus, and apparently undergoing softening. Dysphagia acute, and much pain on deglutition felt inside left ear. Great relief from scarification of epiglottis, and could afterwards swallow two wineglassfuls of milk and sherry without trouble.

57.—M., æt. 47.

Lungs.—Left, dulness and cavern under sterno-clavicular articulation; right, loud murmur and vocal resonance.

Larynx, 9th June.—Pharynx red; epiglottis swollen. 21st July.—Epiglottis pendulous and tubercular; pain in larynx and difficult deglutition; voice hoarse, and at times aphonia. 23rd June.—Throat better, deglutition easier, and less hoarseness.

58.—M., æt. 43, butcher's assistant.

Lungs, Dec. 28th, 1870.—Left, front, no murmur, indistinct friction sounds, no obvious dulness back, low indistinct humid crepitation; right, respiration harsh at base. Three or four attacks of hæmoptysis in streaks; coughs and expectorates much; much dyspnœa. Phthisis not confirmed, though probable; at all events, congestion of lungs. Great pulmonary and laryngeal exertion in calling out weight of meat from the scale.

Larynx, June 1st.—Cannot see vocal cords; indeed, nothing but mere edges or rudiments of true cords to be discerned. Is very hoarse. Probable case of simple congestion of vocal cords.

59.—F., æt. 28.

Lungs, July 7th, 1870.—Left, shallow respiration, dulness, with crepitation; right, respiration deficient and bronchial.

Larynx, 22nd Sept., 1870.—Larynx very red and swollen.

60.—M., æt. 30.

Lungs, Sept. 20th, 1870.—Left and right, fine crepitus, no dul

Larynx, Sept. 20th, 1870.—Arytenoid bodies swollen and i tubercular matter. On attempting phonation, incomplete adc lost their lustre, and appeared uneven. Ill for eighteen months,

twelve months ago. On first applying, aphonia was very complete. In December, 1870, voice improved, but feeble.

61.—F., æt. 17.

Lungs, June 9th, 1870.—Left, dull, loud, cavernous respiration; right, vocal resonance.

Larynx, 9th June, 1870.—Pharynx red; epiglottis swollen; pain in larynx and dysphagia; voice hoarse at the time. 23rd June.—Throat better; deglutition improved. 21st July.—Epiglottis pendulous and tubercular. Iodine inhalation, syrup of iodide of iron, and chlorate of potash.

62.—M., æt. 20.

Lungs, Oct. 12 (1869).—Left, crackling and dull. 24th March, 1870.—Left, dull, no cavity, or crepitation, or click; right, murmur deficient, but otherwise fair. Hæmoptysis in streaks before applying. Brother died of phthisis.

Larynx, 9th Nov., 1869.—Larnx very red; arytenoid bodies and folds swollen and red; voice weak and hoarse. 23rd Nov.—Voice much better, and could also sing. 30th Nov.—Improvement continued. March 24th, 1870.—Apparently no return of the laryngeal affection.

63.—M., æt. 25.

Lungs, Nov. 3rd, 1868.—Left, no dulness, but dry crepitation, very distinct; right, crepitation merging into crackling, no dulness, several severe attacks of hæmoptysis. Nov., 1869.—Left, respiration harsh; right, slight crackling, dyspnœa.

Larynx, Nov., 1868.—Transparent mucous secretion over arytenoid bodies, and false cords; no granular or red appearance; parts rather paler than usual; weakness of the tensor muscles. Nov., 1869.—No laryngeal symptoms.

64.—M., æt. 19.

Lungs.—Left, humid crepitation, murmur very deficient, no dulness; right, loud bronchial wheezing.

Larynx.—Vocal cords could not be seen owing to the congested state of the larynx; epiglottis very red and congested (simple laryngitis, probably passing into laryngeal phthisis).

65.—F., æt. 32.

Lungs.—Very fine crepitation, right; left, normal.

Larynx.—Tubercular spiculæ (follicles) on posterior wall of pharynx, which is thickened; deficient power in adduction of cords, which appeared also relaxed.

66.—M., æt. 33.

Lungs.—Left, very deficient murmur and coarse crepitation; right, a large dry cavity. In ill health for 12 months. No hereditary predisposition to phthisis.

Larynx, March 21.—Pendulous epiglottis; arytenoid bodies very much swollen; aphonia nearly complete.

67.—M., æt. 33.

Lungs.—Crepitus and cavity right apex.

Larynx.—False cords red and swollen; true cords appeared red and had lost their lustre; voice very weak, with a tendency to fall into a whisper.

68.—M., æt. 38.

Lungs, 2nd Feb., 1871.—Some harshness; no expansion or murmur left base; never any hæmoptysis. Mother and two sisters died of phthisis.

Larynx, 2nd Feb., 1871.—Right side of larynx very much swollen, and right vocal cord not visible; left side of larynx congested; aphonia.

69.—F., æt. 37.

Lungs, May 11th, 1871.—Left, some fine crepitus and prolonged expiration; right, respiration fair, though harsh.

Larynx.—Epiglottis pendulous, much swollen, and tubercular; no syphilitic taint; softening apparently in progress on the posterior wall of pharynx (a clear case of laryngeal phthisis, although the physical signs of the chest were still uncertain).

70.—F., æt. 28.

Lungs, Sept. 2nd, 1869.—Right, dull, and bronchial respiration. Oct. 10th, 1869.—Decided large, dry cavity, right apex. Jan. 6th, 1870.—Left apex, a large, dry cavity; right, dry cavity to third rib, sinking in at apex. Hæmoptysis in September, 1869.

Larynx, 28th Nov., 1869.—Both arytenoid bodies were swollen, the left least so; vocal cords healthy. 6th Jan.—Vocal cords healthy, but a raised follicle distinctly seen at back of larynx below the arytenoid bodies; no pain or inconvenience in the larynx beyond a frequent need to clear the throat; the follicles at base of the tongue swollen and red; she thought her voice had been hoarse with occasional aphonia for about ten years. 20th Jan., 1870.—Felt nothing now amiss with the throat; voice quite natural, though perhaps slightly cracked.

URINARY CRYSTALS AND CALCULI:

BEING

OBSERVATIONS ON SOME OF THE CIRCUMSTANCES DETERMINING THE FORMS OF CRYSTALLINE DEPOSITS IN URINE,

AND

ON SOME OF THE CONDITIONS UNDER WHICH RENAL AND VESICAL CALCULI ARE PRODUCED.

BY

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To Mr. George Rainey, my former teacher and deeply respected friend, are owed the inspirations which have led to this and similar investigations. His perfectly original observations on molecular coalescence constitute, as I believe, a discovery the value of which will be abundantly proved in time to come. In the present memoir I record some results of a pathological extension of his physical and developmental researches.

The starting-point of the observations and inferences recorded in this paper is that uric acid assumes remarkably different forms accordingly as it is deposited pure from solution in distilled water on the one hand, or coloured and impure from urine on the other. I beg, leave, therefore,

in the outset to remind the Society of the general nature of this difference.

If some of the colourless or nearly colourless uric acid prepared from guano or serpent's urine be boiled in distilled water, and, after filtration, be left to crystallise from solution, the acid will be found in tabular crystals. Some of the tablets will be oblong in outline, and about six times as long as thick; such tablets, being perfectly transparent and homogeneous, may be compared to the oblong pieces of glass sold as paper-weights; they are the "oblong-square plates" of Dr. Thudichum.[1] Others will be much thinner, much longer, six-sided in outline, with acute points, and may be compared to the common form of glass hand-plates on doors. There will be a number of intermediate forms connecting these extremes. It will be important to notice that the tablets remain generally separate and distinct from one another, or, if here and there aggregated, do not form regular star-shaped masses of crystals radiating from a common centre.

Uric acid deposited from urine differs from the pure uric acid in colour, form, and aggregation.

The crystals are always of a strong yellow or brownish colour, and are most commonly rhombohedra or modifications of the rhombohedron in which that form is easily made out. Prout ('Stomach and Urinary Diseases,' 1840) gives two figures of uric acid. "The first represents," to use his words, "the ordinary appearance of lithic acid crystals under the microscope. The crystals are rhomboidal prisms, thin, transparent, and of a yellow colour." Fig. 2 represents "rhomboidal prisms of lithic acid, thicker than those depicted in the preceding figure, and, in some instances, approaching to the cubic form. The cubic is much more rare than the rhomboidal variety of crystal." There is here no mention of the tabular forms. Dr. Beale, in his well-known work on 'Urinary Deposits,' describes or figures more than thirty forms of uric acid. The edition of 1869 does not contain

[1] 'Pathology of the Urine,' 1858, p. 80.

one drawing of the perfect oblong tablets and only one of hexagonal crystals. A descriptive note beneath the latter points out that this form occurs in urine very rarely.

There must exist, therefore, in urine causes leading to a complete turning away of uric acid from its pure crystalline form. The change, too, is in a definite direction. The faces and angles of a crystal of urinary uric acid are almost always curved and rounded. Such a crystal is clearly the resultant of the operation of two distinct influences,—of crystalline polarity, under which flat surfaces and sharp angles are determined, and of molecular coalescence, in which polarity is lost and particles are arranged in spherical masses by virtue of their unhindered mutual attractions. Furthermore, in urine the crystals are very frequently gathered into larger glomeruli, to which such names as "gravel" and "cayenne pepper grains" are given. These, on examination, are found to be regularly constructed of radiating rhombs or prisms. The difference, therefore, between the *urinary* acid and the *pure* is very pronounced. So also is the difference among the forms of urinary acid. From Prout's two figures the record has gone on growing till as many as forty or fifty distinct varieties can be reckoned. Dr. Miller, Dr. Beale, and other writers on micrology and chemistry have suggested that the substances associated with uric acid in urine are the determining causes of the several differences; and the original observations of Mr. Rainey on the influence of colloids upon crystalline form—observations recently confirmed in a remarkable way by Harting's independent researches—have pointed the way to a solution of the problem. In the 'St. Thomas's Hospital Reports' for the year 1870 I have published an account of a number of experiments and observations which led me to the following general conclusions:

1. That, in the presence of albumen and other colloids, uric acid was deposited in small, thick, subcubical crystals with much-curved faces, or in some kind of dumb-bell, or

in some kind of spheroid. The forms plainly tended to sphericity.

2. That, in the presence of grape sugar and other crystalloids, the acid was deposited in large tabulate or foliaceous crystals with flat sides and sharp edges.

The first experiments were made with artificial solutions from which, in each case, everything but pure acid and one associate substance was excluded; but at the end of the paper a few drawings were given of forms of uric acid taken from albuminous and sugary urine respectively. The figures confirmed in a general way the principle obtained in the exclusive experiments, but left evidently much to be explained.

The present communication relates the results of further investigations, affecting not only uric acid, but also urate of ammonia, oxalate of lime, and triple phosphate.

I. It appeared to me that one of the first subjects of inquiry should be the cause or causes of the difference between the pure and the urinary form of uric acid. Four groups of substances to which determining influence might probably be referred were to be found in urine,—mucus, urea and extractive, colouring matters, and salts. Mucus among these seemed most likely to mould uric acid. Mucus is a colloid, prone to decomposition, and active in promoting the decomposition of organic substances associated with it in solution. On the other hand, mucus in its fresh state is not actually held in solution in the urine, but merely suspended in a very much gelatinised condition. The first experiment consisted in removing the mucus from urine and observing the crystals of uric acid deposited after such removal. Absolute alcohol was added in equal bulk to fresh urine, of specific gravity 1020, clear, of a light sherry colour, and free from sugar or albumen. After standing some time the mixture was filtered, the mucus and some of the salts being thus removed from the liquid, which was afterwards heated to expel the alcohol. After all the alcohol was driven off, the remaining liquid was diluted with distilled water till the specific gravity of 1020 was restored.

The liquid was next divided into four portions. To one a few drops of hydrochloric acid were added; in a second a few grains of pure uric acid were dissolved by the aid of heat; in a third and fourth washed uric acid from urine was dissolved, with and without the addition of two drops of glacial acetic acid. The four solutions were clear, and possessed the characteristic colour and smell of urine. Crystals were obtained from all four; they were in each case small rhombohedra with rounded angles, very much like those represented in Prout's first figure. To complete the experiment, the precipitate of mucus, &c., remaining upon the filter was washed with distilled water till the filtrate was colourless. The precipitate was then washed off the filter-paper and digested in three ounces of distilled water with three drops of hydrochloric acid; one ounce of a solution of pure uric acid in boiling water was added. The crystals deposited from this mixture were small; they were partly thick rhombohedra and partly short four-sided prisms with pointed ends, having therefore often a six-sided outline. They were not aggregated in any way, and were not adherent to the vessel.

The removal of the mucus from the urine, therefore, did not decidedly affect the form of the individual crystals. But their mode of deposit was different from the mode of deposit of uric acid from natural urine. They were caked on the sides of the vessel, and had to be detached for examination, and they were not in spherical radiating masses. Uric acid deposited from urine is rarely caked in this way, and is only in part attached to the side of the vessel. It usually lies in a loose powder among the mucus at the bottom of the vessel, and it is most commonly in radiating masses. The mucus probably arrests, by mechanical opposition, the fall of the crystals as they are formed in the solution, and places them in a favorable position for the addition of fresh crystalline matter on all sides of them. The spherical form of the crystalline masses indicates, I believe, more than a mechanical influence; indicates the molecule-disturbing influence of a colloid. [1]

complementary experiment proves that the mucus does, in fact, affect the form of the crystals, and that the affection is like that of other colloids. But the necessary processes of the experiment cook the mucus, and tend decidedly to weaken its power. It is no more the same mucus that works in natural urine than boiled salt beef is living flesh. It is less putrefiable than fresh mucus, and has therefore less energy in transformation. If, thus weakened, it is capable of transforming uric acid, its natural influence must be considerable.

Supposing the uric acid of human urine and the uric acid having another source to be the same substance, the experiments showed conclusively that mucus with earthy phosphates, &c., was capable of impressing upon it the rhombohedral form, and that urine deprived of its mucus and earthy phosphates had the same power. It was necessary to pass to further exclusions.

Uric acid spontaneously deposited from urine was separated from the other constituents of urine by decantation and repeated washings. It was then thrown upon a filter and washed with distilled water containing a little acetic acid. There would remain then on the filter the uric acid, with perhaps a trace of mucus and epithelium. The prevailing forms of crystal in the uric acid used are drawn in fig. 2. Boiling distilled water was poured upon the filter, and the filtrate was collected and cooled. The solution was of a golden sherry colour, and yielded a large crop of yellow crystals.

Four further recrystallizations were made, each time with loss of colour, and the forms accompanying each loss of colour were noted and drawn (figs. 3, 4, 5 and 6). In the last all colour had disappeared, and the tabular forms of the pure acid were obtained in great beauty.[1] Separate

[1] Uric acid and urates are notable among urinary deposits for carrying down with them the colouring matter. They seem to take the dye much as animal fibres take dyes; and uric acid at least appears to be changed in its crystalline polarities by the dye in a degree corresponding to the amount of dye present with it in solution.

experiments were afterwards made with the various salts of urine. The influence of urea was known before to be in favour of the tablets, and the salts made little or no difference either way. The possibility of some of the constituents of the extractive combining with uric acid and modifying its form had occurred to me; but the experiments noted, and the fact that the form of the acid is the same when deposited in the presence of considerable excess of other acids, were against the supposition.

The final conclusion was that mucus and colouring matter were certainly both agents in determining the ordinary urinary form of uric acid, and that they were probably the sole agents.

ii. *Forms of uric acid in albuminous urine.*—These are always different from the forms found in ordinary urine, but they differ remarkably among themselves. They are small, rounded, and compact, presenting many gradations from the subcubical crystal with curved sides, such as is drawn in Prout's second figure, to the sphere. I have not been able to trace any connection between the variations of form and variations in the quantity of albumen present. Neither have I any observations recording the influence of isomeric varieties of albumen. Urea, however, does appear to be one among the causes of variation. Urea alone, in solutions of specific gravity less than 1040, promotes the formation of large, very thin plates of uric acid, and numerous observations indicate that a large proportionate quantity of urea in albuminous urine constitutes *pro ratâ* an obstacle to the sphere-forming process.

Thus, in albuminous urine of specific gravity 1027 or upwards, yielding a free precipitate of nitrate of urea, on the addition of an equal bulk of nitric acid and subsequent cooling to 60° Fahr., I have constantly found the forms shown in the adjoined figures (fig. 7). These have been drawn for me by Dr. Donkin, who was kind enough to help me in a series of observations made at St. Thomas's Hospital. They may be arranged in three groups:—

1. Small, thick, sharp-edged rhombohedra; almost cubical.

2. Rhombohedra having the obtuse angles rounded, and the axis joining the flat sides lengthened, whereby a long cylinder with flat ends is produced. (Compare fig. 3, p. 339, 'St. Thomas's Hospital Reports,' 1870.)

3. Rhombohedra having the margins of the flat sides swollen and rounded, the crystal between being thinned and marked with diagonal lines. (Compare fig. 5, a, d, and e, at p. 340 of the 'Reports,' above quoted).

Secondly, with a lower specific gravity (1015 to 1021) the forms drawn in fig. 8 by Dr. Donkin, and in fig. 9 by myself, occur. The cylinders are thicker and shorter.

Lastly, in albuminous urine of still lower specific gravity (1009 to 1015) there are found, associated with very small rhombohedra, tub- or cask-shaped crystals (doliola). The rhombohedron is still to be detected under the disguise of the doliolum. The alternate obtuse and acute angles are rounded off, making the section parallel to the flat end-faces first lozenge-shaped, then broadly ovate, then circular. A deeply shaded line frequently runs like a hoop across the middle of the cask, and this is again bisected by faint diagonal markings. The ends of the doliolum are rarely quite flat. Sometimes they are excavated between the diagonals, and become a double cup (ἀμφικύπελλον) like the wooden egg-cups to be seen in some country inns. Sometimes the head of the cask is projected into a bundle of spicular crystals, the substance of the cask giving at the same time indications of the formation of longitudinal lines of crystallisation (fig. 10). Sometimes the cask is so short and its ends are so rounded that it has become a sphere with a long spike projecting from each end. The perfect spherical form of uric acid is not often found in urine; the dumb-bell is even more rare. I have, however, found both in albuminous urine of very low specific gravity. The spheres are always very small and homogeneous when deposited in urine. But in experiments made with watery solutions of egg-albumen I have obtained large and beau-

tiful spheres, some equalling fat-cells in size and displaying a briliant radiating crystalline structure. I conclude that even in small quantities urea still hinders the completion of the collospheres of uric acid. Urea is known to modify by its presence the form of the crystals of chloride of sodium, and its influence may be in part merely such an influence as we have seen it exerting on uric acid in exclusive experiments. It may also, like saline matter in solution, retard the decompositions of albumen.

iii. *Forms of uric acid in sugary urine.*—Here the crystals tend to be tabular. Whereas with albumen the acute angles grow larger and the obtuse angles smaller till equality is attained and the cube prevails, with sugar the obtuseness of obtuse angles and the acuteness of acute angles are increased; whereas with albumen the axis joining the flat sides of the rhombohedra is lengthened, with sugar that axis is shortened. Two forms of uric acid may be noted in connection with sugar in urine; one oblong or square in outline, with fine chiselled ends and thicker middle; the other much less common, with flat parallel surfaces and diamond-shaped or hexagonal outline. Drawings of both are annexed (figs. 11 and 12).

iv. *Forms of uric acid in purulent mucus.* — Colloidal substances allied in composition or reactions to purulent mucus contribute, if my observations are correct, to the formation of stone in the kidney and bladder. The urine of chronic cystitis, abounding in purulent mucus, is mostly advanced in decomposition far enough to have become ammoniacal, and filled with precipitated phosphates at the time when examination has been possible. Urine of this kind, secured early, and, after gentle warning, treated with a few drops of hydrochloric acid, gives very interesting forms. In the fluid portion of such urine rhombs and rosettes are formed just as in natural urine. In the mucus, cubes and rounded bodies and dense stalactitic agglomerations of uric acid are deposited, the precipitate within the mucus being much larger than in the surround-

ing fluid. Artificial conjunctions show very clearly that the actively changing colloids entering into the matter of purulent mucus exert a strong converting and cementing influence on uric acid. For instance, the stringy sediment from some cystitic urine was separated on a filter, and treated successively with liquor potassæ, hydrochloric acid, and distilled water, until no crystalline forms could be seen under the microscope. The residue, flaky and much less cohesive than before, was heated in a solution of pure uric acid. The liquor being cooled was found to be full of six-sided and long thin halbert-shaped crystals. The mucus, where thin and but little cohesive, contained small subcubical and rounded forms; where thicker and more gummy it contained the same forms, with the addition of a stalactitic and columnar growth of large irregular crystals with rounded margins. These sticky flakes were hard and gritty when compressed between the fingers, and were practically the beginnings of calculi formed under one's eye. The larger columnar crystals, resembling nothing that I had seen in urine before, find their counter-part in a drawing by Dr. Vandyke Carter, of "crystals forming columns," from a urethral calculus. This drawing occurs in fig. 5 of Plate I, illustrating Dr. Carter's valuable book on the "Microscopic Structure and Mode of Formation of Urinary Calculi." The main point demonstrated in that book by Dr. Carter is, that in calculi the crystalloids are found in spheroidal forms, or in forms tending to be spheroidal associated with a colloid bed. The form and the cohesion of the crystalloids are explained according to the principles of Mr. Rainey's theory of molecular coalescence. Dr. Carter suggests that "an excess of mucus, perhaps altered in character, in the urinary passages, or the effusion of albumen, fibrin, or blood and the like, say from congestion of the kidneys, or from irritation of the urinary tract, would furnish a colloid medium with which uric acid, the urates or oxalates, themselves perhaps in excess would combine in the manner before described." Mucus in particular commends itself to Dr. Carter as a colloid most

likely to be actively concerned in the formation of calculi. The purulent mucus poured out in irritation of the urinary passages should certainly be just the substance to effect the formation of calculi, if the inferences which I have elsewhere drawn from the observed influence of colloids upon oxalate of lime and other salts are correct.[1] A fact upon which I was led to lay great stress was, that whereas oxalate of lime was turned into dumb-bells and spheroids by gelatin, the conversion was much more active and complete where the gelatin was undergoing decomposition and when the temperature was raised. In experiments I have not had opportunities of observing uric acid deposited at the temperature of the body. But it is of course quite certain that under particular circumstances uric acid is frequently deposited in the urinary passages, and is therefore deposited at temperatures ranging near 100° Fahr. I entertain no doubt whatever that if uric acid in the act of crystallisation, or during its stage in the body after crystallisation, meet, at such temperature, with purulent mucus in full activity of molecular change, the formation of calculous bodies, more compact than those which I have been able to obtain outside the body at ordinary atmospheric temperatures, will follow. Two thirds of all urinary calculi are in bulk composed of or start from concretions of uric acid. Writers on the stone have, therefore, directed their attention to the causes of the formation and deposit of uric acid as the most important considerations in explaining the occurrence of calculi, but if Dr. Carter's examinations of calculi and my experiments, here and elsewhere related, have any value, it is necessary to take into account besides the deposit of uric acid the existence of conditions sending colloids into the urine. The pebbles of the concrete would not hold together without the cement to bind them, and act on their surface. To make calculi of uric acid without colloids would be as hopeless a task as making ropes of sea sand. Therefore, if the causes of the varying prevalence of calculus disease in different communities and

[1] 'St. Thos. Hosp. Rep.,' 1871.

regions are to be fully and usefully examined, such subjects as constitutional proneness or indisposition to vesical catarrh, habits of life, diet and regimen, or local conditions of soil, water, and climate which can hinder or promote the secretion of mucus, or the occurrence of irritation in the urinary tract, must be very carefully treated. And if such observations be applied to practice, we may hope to find that the prevention of stone becomes partly possible through the prevention of the local conditions which constitute the soil in which it is sown and grows.

v. *Forms of urates.*—In many cases I have observed small brown spherules, soluble at moderate temperature, in albuminous urine depositing uric acid. They dissolved much more readily than the acid, and were either urate of soda or urate of ammonia.

In ammoniacal urine containing much purulent mucus I have frequently seen urates in three forms: 1, perfect spheres; 2, spheres with regular radiating raphides; 3, spheres with an investment of branched and irregular or bristly raphides ("hedge-hog" crystals) (fig. 15).[1] I have procured these in great abundance by adding to an acid urine containing large quantities of urate of ammonia the stringy mucus from ammoniacal urine of chronic cystitis. The mixture being first warmed to dissolve the urates, they are found, after cooling, deposited in two forms; within the mucus as spheres and hedge-hog crystals, elsewhere as granules. I have also often observed the spherules of urate of ammonia which have been described by Dr. Roberts as formed in decomposing urine, and have no doubt of their being formed under the influence of the chemically-active mucus.

A spherical form of urate of soda was figured, at first under the name of urate of ammonia, by Dr. Golding Bird in his urinary deposits. He described it as follows:—"Spherules of urate of ammonia with crystals of uric acid adhering." And he stated that this form was occasionally observed in albuminous urine occur-

[1] "Thom-apples" of German authors.

ring in scarlatina. Dr. Thudichum, Dr. Beale, Dr. Roberts, and Drs. Ultzmann and Hofmann, have since figured similar forms. There is no further note of albumen, but the associated conditions were usually febrile attacks in children. Drs. Thudichum and Roberts have added a third condition, evidently of much importance—delay of the urine in the urinary passages. In all the cases where any history or explanation is given there have been states favouring the presence of colloids, either of albumen or of mucus, or of other results of rapid febrile action. And delay in the urinary passages, keeping the urates long in contact with putrescent colloids, at a temperature most probably above 100° Fahr., must have added a very strong impulse to the forces determining spherical form.

The urates, though the most common of all deposits from cooled urine, enter much less frequently than uric acid into the composition of calculi. Their comparative solubility, and their habit of assuming a fine molecular (amorphous?) state without running into masses, will account for this. Their solubility has been shown by Dr. Bence Jones[1] to be increased by the presence of chloride of sodium, distilled water being rendered capable of taking up double the quantity of urate of ammonia when the chloride is added in small quantity. The same addition prevents urate of ammonia from taking the crystalline form. Dr. Bence Jones states, that if urine is added to a warm solution of needles of urate of ammonia in such small quantity as not to cause any discoloration of the subsequent deposit, that deposit is always amorphous. He traces this to the chloride of sodium. Whether the colouring matter, which adheres with such tenacity to the urates that it is best obtained from them (Golding Bird, Thudichum), affects the form of urates, I have not as yet determined.

vi. *Oxalate of lime.*—Dr. Beale has figured dumb-bells of oxalate of lime in casts of urinary tubules. His remarks upon these figures, indeed, directed my thoughts to the pre-

[1] 'Trans. Roy. Med.-Chir. Soc.,' 1844, p. 102.

sent investigation. Following up Dr. Beale's[1] hints, I have shown elsewhere that oxalate of lime is much modified in its crystalline form by the presence of colloids, exchanging the octohedron for various tablets, dumb-bells, "wheat-sheaves," and spheres.[2]

I have a few notes of modifications of oxalate of lime in urine. One instance is so remarkable that I must quote it at length. A friend sent me one day a slide of beautifully mounted crystals of oxalate of lime; mixed with variously altered octohedra were multitudes of large lustrous "wheat-sheaves," or crystalline dumb-bells. He told me that they had been mounted by the gentleman who had passed them. This gentleman was subject to violent attacks of headache, during which he passed very turbid urine. Being accustomed to make much use of the microscope, he examined the deposit for himself, and found these beautiful crystals. On receiving the specimen I wrote at once to ask if the urine contained albumen or mucus, and was informed that during the attacks the urine did contain albumen, and that it was free from albumen at other times. Dr. Thudichum gives figures of dumb-bells of oxalate of lime from albuminous urine.

I adjoin two more illustrations of altered oxalate from urine. The first (fig. 17) of oxalate taken from slightly acid urine of sp. gr. 1016, containing both albumen and stringy mucus; the second (fig. 18) from neutral urine of sp. gr. 1021, containing much mucus and vaginal epithelium. There was no sugar present in either case.

To mould oxalate of lime into calculi would seem to require denser colloids than are usually present in vesical urine, and it is probable that the beginnings of oxalic calculi take place in the recesses of the kidney among less diluted colloids. Certainly, in my own experience I have found the small calculi passed with so much pain from the kidneys much more frequently composed of oxalate of lime than are vesical and urethral calculi.

[1] 'Urine and Urinary Deposits,' 2nd edit., p. 347, W. S. C.

[2] 'St. Thos. Hosp. Rep.,' 1871.

vii. *Triple phosphate.*—The crystals of this and other phosphates are much less influenced by colloids than the substances with which I have been so far dealing. In urine containing much albumen or purulent mucus I have often found crystals rounded at the angles and altered considerably from their natural beauty. But only a part of the crystals have been so changed, and there has been no observation of spherical or agglomerated forms. By the use of a temperature a little higher than that of the body I have been enabled to effect the moulding of these crystals into calculous masses. If ammoniacal urine of chronic cystitis containing plenty of purulent mucus be digested for twenty-four hours at a temperature ranging between 100° and 110° Fahrenheit, the mucus is afterwards found full of spheroidal forms, many of them very large. The reactions of these indicate them to be in part phosphates, but the presence of urates deprives the experiment of conclusiveness. The mucus must be separated, treated with caustic alkali and hydrochloric acid, and after good washing be placed in a slightly acid solution containing triple phosphate. A small excess of ammonia is added after a while, and the mixture is then digested for twenty-four hours at 110° Fahrenheit. The flaky and much coagulated mucus is found then to contain rosettes of crystals almost identical in form with certain figures given by Dr Vandyke Carter at Plate IV, fig. 2 *b* and. fig. 3 *a*, *b* of his book. Dr. Carter's crystals were from the outer layer of a small pale calculus and from the nucleus of a small pale calculus respectively.

viii. *Influence of albumen on nitrate of urea.*—Nitrate of urea, as is well known, can often be obtained from urine without previous concentration. The crystals thrown down from urine of a specific gravity exceeding 1037 after the addition of an equal bulk of nitric acid are shining flakes, often cohering in irregular masses and distinctly darker than the urine. If urine of such specific gravity contain a moderate quantity of albumen, the addition of an equal bulk of nitric acid causes the solution

of the first precipitate, and a perfectly clear liquid is obtained. The process of crystallization is in this case very different. Instead of dark flakes, tiny globes of a light colour are suddenly seen suspended at several points in the liquid and grow rapidly under the eye. They are composed of fine radiating acicular crystals, and look like little drops of fat. That albumen is the cause of this growth of spheres may be readily proved. Another portion of the fresh urine is freed from albumen by boiling, the addition of two drops of nitric acid, and filtration. The filtrate, treated as before with nitric acid, yields flaky crystals.

ix. *Bearing of the foregoing observations on microscopic morphology.*—Dr. Carter in his remarks on the formation of calculi does not fail to recognise the importance of the higher temperature of the body as a condition of the process. How far this influence is to be attributed to the unchanged movements of heat, and how far to induced molecular changes in the colloids, cannot be decided or calculated at present. Both of these will, of course, come under the head of molecular disturbances, but whether one or other form of molecular movement is more apt to blur the lines of crystallization I do not pretend to suggest. It is only necessary now to point out that heat plays a remarkable part in determining the assumption of spheroidal shape by crystalloids in the presence of colloids. I have already, in the paper referred to, shown this to be the case when oxalate of lime is deposited in albumen. Experiments with phosphate of lime and carbonate of lime in albumen have since confirmed the first inductions.

I have long felt that the present inquiry is only part of a much larger questioning of nature—of an inquiry into the causes, general and particular, by which the morphology of tissues is determined. Microscopists have been content for the most part to record the forms of the tissues with constantly increasing research and accuracy. This is undoubtedly a work of importance, indeed of necessity, but is, to

my mind, only a preliminary process, bearing to the higher morphology of the future the sort of relation borne by the Linnæan to the Natural system in Botany. There must follow a natural system, interpreting the meanings of forms, compelling form to tell the story of growth and function, recording the relations between form and substance, and proceeding to the discovery of the laws of tissue formation. The line of thought into which my observations have directed me leads to the belief that such further research will put us in possession of new methods in diagnosis and therapeutics.

DESCRIPTION OF PLATES V AND VI.

PLATE V.

FIG. 1.—Purified uric acid. Deposited from solution in boiling water. The forms are the same with and without the addition of acetic acid. Experiment.

FIG. 2.—Uric acid, from urine of sp. gr. 1030, containing mucus and much coloring matter, but no albumen or sugar. Observation.

FIG. 3.—The same uric acid separated with the mucus by filtration, and precipitated from solution in boiling water. Exp.

FIG. 4.—The same, second and third boilings; less colour. Exp.

FIG. 5.—The same, fourth boiling; still less colour. Exp.

FIG. 6.—Fifth boiling; no colour. Exp.

FIG. 7.—Uric acid from albuminous urine of sp. gr. 1027 and upwards (Dr. Donkin). Obs.

FIG. 8.—Uric acid, from albuminous urine of sp. gr. 1015 to 1021 (Dr. Donkin). Obs.

FIG. 9.—Uric acid, from albuminous urine of sp. gr. 1015 to 1021 (W. M. O.). Obs.

FIG. 10.—Uric acid, from albuminous urine of sp. gr. 1009 to 1015 (Dr. Donkin and W. M. O.). Obs.

FIG. 11.—Uric acid, from sugary urine, sp. gr. 1042; no albumen; not much colour. Obs.

FIG. 12.—Uric acid, from sugary urine, sp. gr. 1023; no albumen; very acid. Obs.

PLATE VI.

FIG. 13.—Uric acid.—In watery fluid round mucus. Experiment.

FIG. 14.—Uric acid..—In mucus. Exp.

FIG. 15.—Urates (probably urate of ammonia) in purulent mucus. Obs.

FIG. 16.—Oxalate of lime, from albuminous urine. Obs.

FIG. 17.—Oxalate of lime, from urine containing much mucus; acid; sp. gr. 1016. Obs.

FIG. 18.—Oxalate of lime, from urine containing much mucus and vaginal discharge; neutral; sp. gr. 1021. Obs.

FIG. 19.—Triple phosphate. Deposited in flaky mucus at temperature of 100° to 110° Fahr. Exp.

Plate V.

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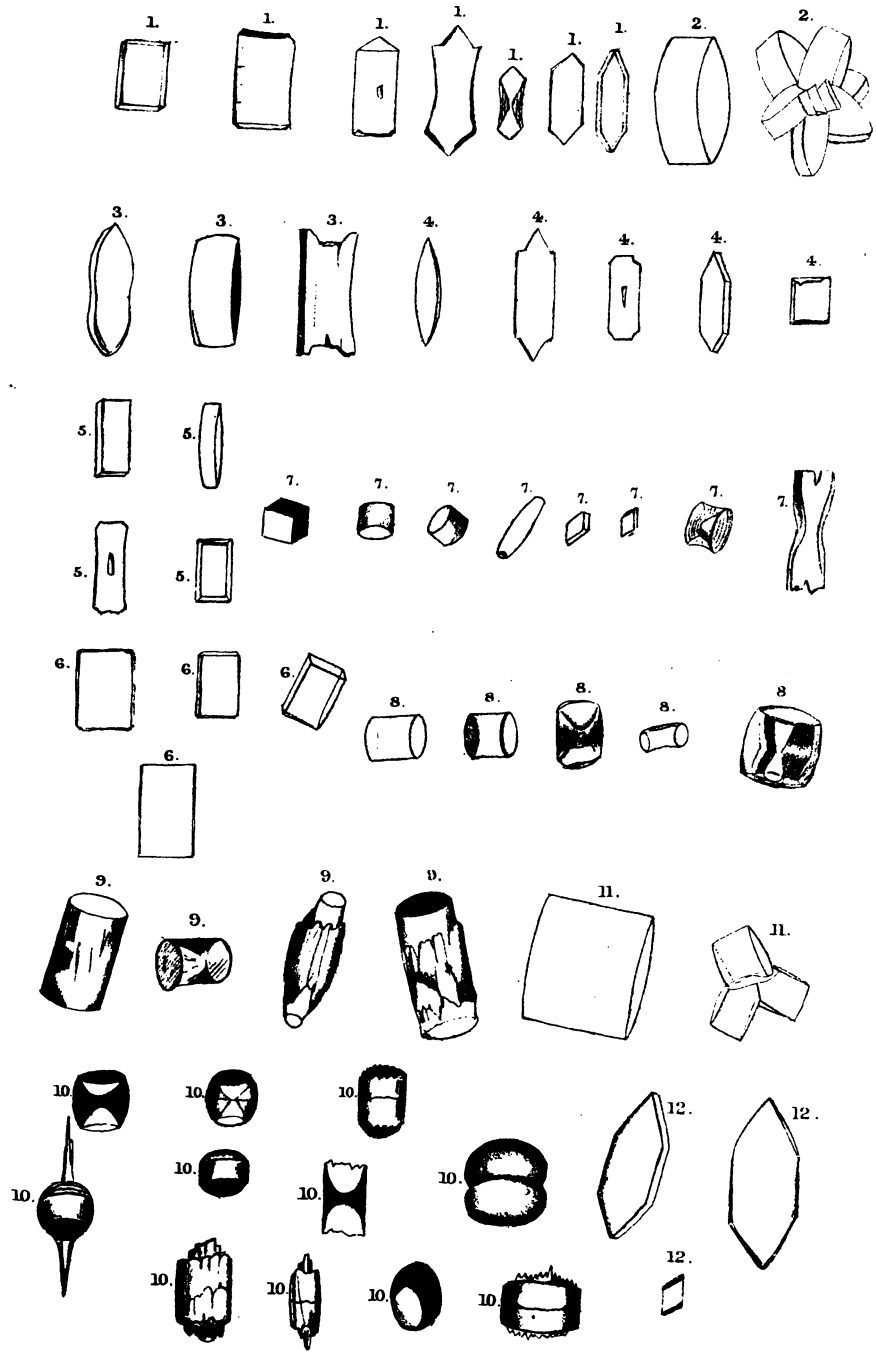
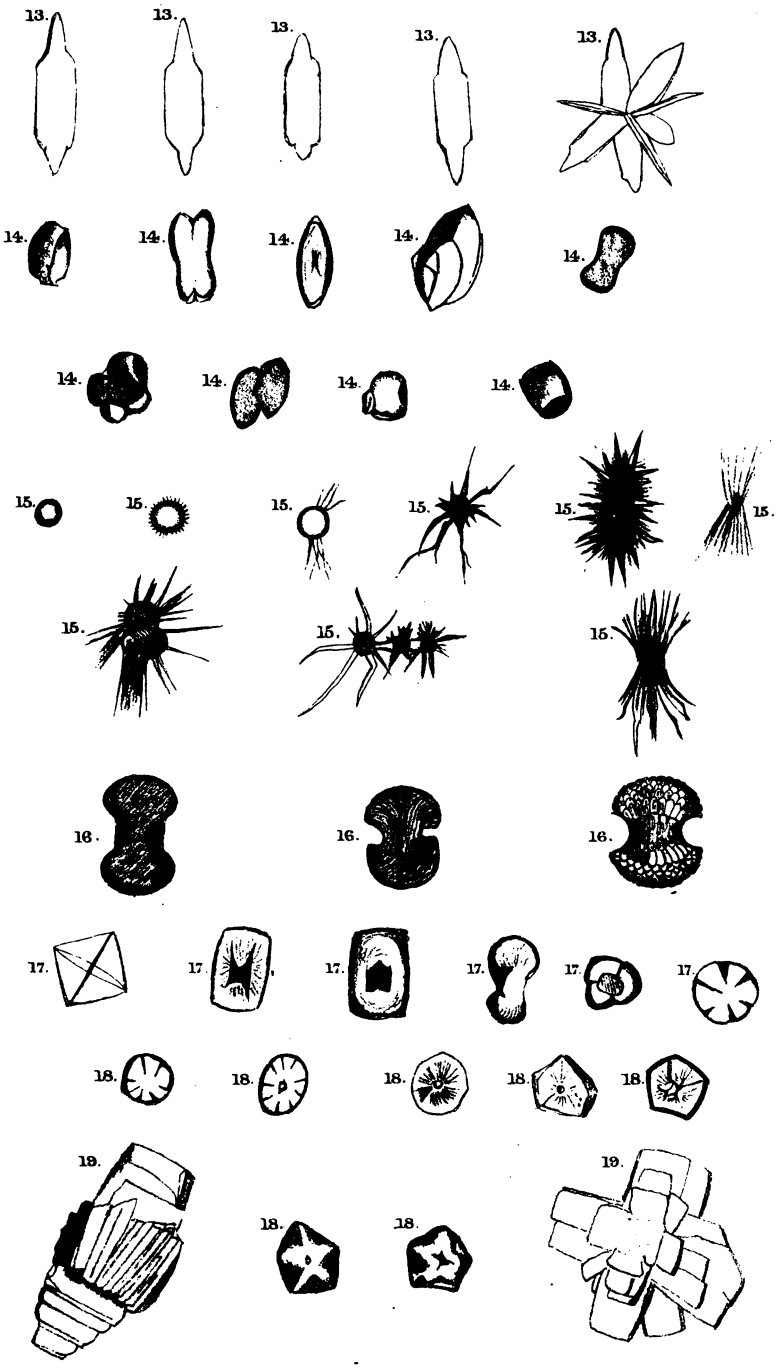


W.M.O. del. G.H.Ford lith.

Mintern Bros. imp.



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A CASE

OF

PULSATING TUMOUR OF THE LEFT ORBIT,

CONSEQUENT UPON A FRACTURE OF THE BASE OF THE SKULL,

CURED BY LIGATURE OF THE LEFT COMMON CAROTID ARTERY SUBSEQUENTLY TO INJECTION OF PERCHLORIDE OF IRON AFTER DIGITAL COMPRESSION AND OTHER MEANS OF TREATMENT HAD FAILED.

WITH

REMARKS AND AN APPENDIX CONTAINING A CHRONOLOGICAL *RÉSUMÉ* OF RECORDED CASES OF INTRA-ORBITAL ANEURISM.

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BEFORE narrating the case which forms the basis of the present paper, it seems desirable to give a brief sketch of the views and observations which previous authors have placed on record concerning the affection which is now generally termed, but with doubtful propriety, Intra-orbital Aneurism.

On the 1st of November, 1809, a paper by Mr. Travers was read at this Society, with the title, "A Case of Aneurism by Anastomosis in the Orbit cured by the Ligature of the Common Carotid Artery." This is generally believed to be the first instance of intra-orbital aneurism of which any

record exists, and it was described by Mr. Travers as aneurism by anastomosis, because he recognised in it a strong resemblance to some of the cases of that affection which had been brought under the notice of the profession by John Bell not many years before.[1] In 1812 Mr. Dalrymple communicated to this Society the second observation in a paper which, following the views of Mr. Travers, he headed, "A Case of Aneurism by Anastomosis in the Left Orbit cured by tying the Common Trunk of the Left Carotid Artery."

In 1823 Mr. Guthrie, without questioning the pathology of his predecessors, recorded, in his 'Operative Surgery of the Eye,' with unfortunate brevity, a case in which symptoms existed on both sides resembling those exhibited unilaterally in the cases of Mr. Travers and Mr. Dalrymple, and in which he found after death, *in each orbit, an aneurism seated on the ophthalmic artery.*

M. Roux's partially successful case, which occurred in 1829, appears to have been only incidentally recorded at the time, and to have escaped the notice of observers in this country until it was reproduced nearly thirty years later in the pages of M. Demarquay. Warren's successful case of ligature of the common carotid for a similar affection after

[1] Mr. Travers states that his case bore also a strong resemblance to the case "communicated by Mr. Freer, of Birmingham, whose patient, refusing assistance, expired of hæmorrhage" ('Med.-Chir. Trans.,' vol. ii). At the present time it is difficult to recognise a likeness between Travers's case and that of Freer. The latter ran its course in a few months, the eye protruding and suppurating within a few days, whilst Travers's case had commenced more than four years previously to his seeing it, and had not made any rapid progress. Middlemore also places Freer's case under the head of "aneurism by anastomosis." But Freer himself described his case as one of "fungus hæmatoides," and he figures a growth as large as an adult fist sprouting from the left eye. Mr. Nunneley also regarded Mr. Freer's case as one of fungus, and believed that it was to Freer's case that Middlemore alluded in the following passage:—"I have only seen this affection once, and then the patient was not under my own care; no operation was performed for his cure, and he died, as I think, in consequence of the omission of that assistance which the improved state of surgery has placed us in a condition to bestow on persons suffering from this most distressing disease." (Middlemore on 'Diseases of the Eye,' vol. ii, p. 618.) Compare with Freer's case, plate 18 in 'Heister's Surgery,' vol. i, p. 431.

failure of local treatment occurred in 1830, and was published in 1837 as a case of aneurism by anastomosis. "In the same year, at a period previous to the first operation," Warren had met with a case of pulsating tumour of the orbit of traumatic origin, and had "tied the carotid artery without an alleviation of the disease." In 1839 Mr. Busk communicated to this Society the account of a case under his care in 1835, which had resulted from a fracture of the base of the skull, and which he had cured at the beginning of 1836 by tying the common carotid artery. Mr. Busk added the prior case of a boy under the care of Mr. Scott in 1834 at the London Hospital, in whom protrusion and pulsation of the eyeball supervened after an injury to the head occasioned by a fall into a ship's hold. Five weeks later, the occurrence of profuse arterial hæmorrhage from the nose rendered it necessary for Mr. Scott immediately to apply a ligature to the right common carotid artery. Pulsation ceased, the eye retired into the orbit, but vision previously lost was not regained. Mr. Busk's paper embodied some forcible arguments against the generally received view that the cases of Travers and Dalrymple were examples of aneurism by anastomosis, and, adducing the observation of Guthrie, advanced the opinion that his own case, and most likely also the cases of Travers and Dalrymple, were really instances of ordinary aneurisms affecting the ophthalmic artery within the orbit. The paper had a marked influence on the views of subsequent observers, and, whilst many adhered to the pathology of Travers, not a few echoed the arguments of Mr. Busk, and designated their cases as "aneurisms of the ophthalmic artery."[1] It was in 1835 that M. Baron pre-

[1] Through a singular error M. Demarquay affirmed in his treatise on tumours of the orbit that the rupture of the ophthalmic artery diagnosed in Mr. Busk's case had been verified at a later date by an autopsy; whereas Busk's patient was alive and well when he last came under observation. I find the same statement echoed by Dr. Noyes in his table of cases of ligature of the carotid for pulsating tumours of the orbit ('New York Medical Journal,' 1869, p. 666).

Among subsequent observers M. Petrequin, Mr. Curling, M. Bourguet, M. Legouest, Mr. Zachariah Laurence, M. Aubry, Professor Gioppi, Dr. Morton,

sented to the Société Anatomique at Paris a specimen of *aneurism of the internal carotid artery in the cavernous sinus, which appeared to have been ruptured* in that situation, and to this condition he attributed the varicose state of the orbital veins, the exophthalmos, and bellows murmur which had been noted during life. The observation was so brief that it naturally escaped notice, and failed to make any impression on pathological opinion.

In 1839 two well-known cases occurred in Paris, those of Jobert and Velpeau. In Velpeau's case, which was seen by Mr. Erichsen, both orbits were affected with a pulsatile tumour following a blow on the nape, and no less than three different accounts have been given of the effects of compression of the carotid, rendering it difficult to determine which account should be accepted.[1] It is at least certain that the

and Dr. Schmid, of Odessa, diagnosed their cases as examples of aneurism of the ophthalmic artery.

[1] The following are the different accounts which have been given:—Mr. Erichsen ('Science and Art of Surgery,' vol. ii, p. 89) says—"In a very interesting case which I saw in Velpeau's Wards in 1839 both orbits were affected, and as pressure on the right carotid arrested the pulsation and bruit in both, that artery was tied. But though the disease was cured in the left orbit by this operation, and temporarily arrested in the right, it reappeared in the latter situation, and was eventually cured here by the ligature of the left carotid." The statement that the left carotid was tied would appear to be a mistake. Velpeau proposed to tie it, but the patient would not consent (see Delens' 'De la Communication de la Carotide Interne et du Sinus Caverneux,' p. 57). Velpeau himself (in his 'Leçons Orales,' and art. "Orbite" in the Dictionary in thirty volumes) states that pressure on the right carotid arrested completely the bruit and pulsation in the left orbit, allowing them to remain in some degree on the right side, whilst pressure on the left carotid stopped the bruit and pulsation on the right side only. The contemporary account published in the 'Bulletin de Thérapeutique' of 1839 does not sustain the account of the *crossed effects of compression of the carotids* so strongly insisted on by Velpeau. It merely states that the bruit ceased at once on compression of the carotid of the corresponding side, and a most remarkable circumstance was that it ceased on the left side almost as completely as on the right by the sole compression of the right carotid. M. Carron du Villards says — "In the patient operated on by M. Velpeau, who was affected with two exorbitisms produced by a presumed rupture of the two ophthalmic or orbital arteries, compression of the right carotid suspended the pulsation in the two orbits; whilst that exercised on the left carotid produced no result, even in the corresponding orbit"

effect of ligature of the right carotid practised by Velpeau was the immediate cessation of pulsation and bruit on both sides. At the end of three months the symptoms returned on the right side; and as pressure on the left carotid caused them to cease entirely, Velpeau recommended ligature of that artery, but the patient would not consent. The cure remained permanent on the left side. Velpeau mentions a second case which occurred about the same time, but it does not appear to have been recorded. All that Velpeau says of it is, that the affection was on one side only, and that the patient referred it to a blow on the nape.[1]

M. Gendrin published in 1841 the account of a remarkable case which he met with in 1835, and in which the post-mortem appearances point to the *rupture of an aneurism of the internal carotid in the cavernous sinus.* Dr. Dudley, of Lexington, tied the common carotid in 1841, and M. Herpin in 1844, for pulsating tumours in the orbit.[2] In none of the previous cases which had been submitted to ligature, eleven in number, had a fatal result occurred, but this series of successes was broken by M. Petrequin's case in 1845. Ligature failing, galvano-puncture was tried without avail. Unfortunately no post-mortem examination could be made. In 1851 Gervasi cured a case described as aneurism by anastomosis of the right eye by ligaturing the carotid. Mr. Haynes Walton tied the right common carotid artery successfully in a girl, aged four months and three weeks, for an affection of the right orbit, which was considered by all the surgeons who saw it as a case of "aneurism by anastomosis,"[3] and Brainard cured a pulsating tumour which had returned after ligature of the carotid by

('Annales d'Oculistique,' 1858, tome xl, p. 126). This last account is readily explicable on the supposition of a communication between the right carotid and the cavernous sinus; the other accounts are not so intelligible, but it is of little use to speculate on the subject (as Dr. Delens does) in the uncertainty produced by different statements.

[1] 'Leçons Orales,' t. iii, p. 437.

[2] Mackenzie cites Dr. Dudley's case as an example of true aneurism. Demarquay regards it as an instance of aneurism within the orbit, which had become diffuse by rupture of the aneurismal sac.

[3] Mr. Walton referred to the cases of Travers, Dalrymple, and Warren, as

injecting a solution of lactate of iron. Then followed the first case of Mr. Nunneley, and the case of Mr. France, Ophthalmic Surgeon to Guy's Hospital. In 1853 M. Aubry met with a remarkable case, not published till 1864, in which all the symptoms of an intra-orbital aneurism existed, and which he had an opportunity of examining after death. By injecting the arteries and dissecting them he ascertained that there was *no arterial lesion whatever, but the ophthalmic vein and its branches were enlarged, and had formed the bossy vascular pulsating tumours observed during life. The ophthalmic vein was as large as the little finger, with very thin walls. The cavernous sinus was greatly dilated, and freely communicated with the varicose ophthalmic vein, but terminated behind in a cul-de-sac, the communication with the inferior petrosal sinus being cut off.*

In 1854 Mr. Curling communicated to this Society a paper entitled, "A Case of Traumatic Aneurism of the Ophthalmic Artery, consequent on Injury to the Head, cured by Ligature of the Common Carotid Artery." Mr. Curling supported the views of Mr. Busk, and conjectured that in his own case the ophthalmic artery had been wounded by a spiculum of bone detached by a fracture of the base of the skull.

Dr. Van Buren also tied the carotid artery successfully in a similar case, and the patient remained well when seen a year and a half afterwards.[1]

Mr. Critchett tied the carotid artery for an affection described at the time as aneurism by anastomosis of the right orbit.

The patient went on uninterruptedly well for several weeks, when attacks of hæmorrhage from the orbit occurred, and resulted in death four months after the operation. In all probability the case was one of malignant disease.

In the following year M. Bourguet cured a traumatic case

instances of aneurism by anastomosis in the orbit, and to those of Guthrie, Scott, and Busk as aneurisms of the ophthalmic artery, Scott's case being regarded as diffuse aneurism ('Oph. Surg.,' p. 258, *et seq.*). In his recent edition he has adopted the views of Mr. Nunneley.

[1] See Dr. Noyes' article, 'New York Med. Journ.,' 1869, p. 664.

by injecting the perchloride of iron after the failure of galvano-puncture; and Mr. Hussey met with a case of doubtful character, the nature of which he was unable to ascertain, because an examination of the body could not be obtained. M. Nélaton's first case, occurring in the same year, marks an important advance in our knowledge of the pathology of the affection. The patient had received a thrust in the *left* lower eyelid from the ferrule of an umbrella, and exhibited all the symptoms of an intra-orbital aneurism on the *right* side. By acuteness of reasoning, and by experiment on the dead body,[1] M. Nélaton diagnosed a wound of the right carotid in the cavernous sinus. The patient died of arterial hæmorrhage from the nose, and the diagnosis was confirmed. The *body of the sphenoid bone was fractured, and the right internal carotid was found torn across in the cavernous sinus. The sphenoidal sinus communicated with the cavernous sinus on the one hand, and the nasal fossæ on the other, and through this communication the hæmorrhage had taken place.*

The year 1856 was signalised by the successful application of digital compression in an idiopathic case by Professor Gioppi, of Padua.[2] Mr. Nunneley's second case of ligature

[1] M. Nélaton, endeavouring to account for the mechanism of the lesion, supposed that the ferrule of the umbrella, penetrating by the wound of the left inferior eyelid, had traversed the body of the sphenoid and wounded the right internal carotid, in the cavernous sinus. He even succeeded in reproducing that injury by driving a spike of wood obliquely across an incision made in the lower eyelid. The extremity of the spike passing across the body of the sphenoid, penetrated into the right cavernous sinus, and there compressed the carotid artery. M. Nélaton therefore concluded that there was an aneurism of the right internal carotid in the cavernous sinus. He excluded the idea of aneurism of the ophthalmic artery because that would have compressed the optic nerve at the level of the optic foramen. M. Nélaton's specimen, showing the wood driven from the left side to the right carotid artery in the cavernous sinus, is preserved in the Musée Dupuytren. Mr. T. Holmes, in a note to his 'Lectures on Aneurism,' observes, "I once assisted Mr. Prescott Hewett in a similar experiment, and with an equally successful result, and I produce a preparation in which a pair of scissors is thrust from the lower eyelid on one side into the cavernous sinus on the other, puncturing the internal carotid. The third nerve has just escaped." ('Lancet, 1873, vol. ii, p. 144.)

[2] Gioppi described his case as an aneurism of the ophthalmic artery, and

occurred in the same year. In 1857 Dr. Gurdon Buck tied the right common carotid artery in a traumatic case, effecting only partial improvement. After his discharge from the hospital in New York the patient went to sea, and coming to England was seen at one of the London hospitals. Returning to Dr. Gurdon Buck's care in February, 1859, he submitted to ligature of the left carotid, and in the course of a few months he got rid of the disease, vision being abolished. This case is regarded by Dr. Noyes as the case which was seen by Mr. Poland at the Moorfields Ophthalmic Hospital.[1] Dr. Buck's case is specially interesting as being the first (excluding McGill's and Ullman's cases) in which both carotids had been tied for the affection under consideration. An interesting case also was that of Dr. Halstead, who tied the left

supported his view by reference to the cases of Guthrie, Busk (whom he called Burk), Scott, Curling, and Bourguet. He cites a pathological observation of Carron du Villards who found accidentally an aneurism of the ophthalmic artery before entering the orbit, as well as the case of Rosas. The cases of Travers, Dalrymple, Roux, Guthrie ("1834"), Velpeau, Jobert, Herpin, Walton, and Brainard, he calls arterial telangeiectases. Triquet, who reported Herpin's case in 1852, is cited by mistake as having had a case of his own (see 'Annales d'Oculistique,' 1858).

[1] Dr. Noyes ('New York Medical Journal,' March, 1869, p. 664) gives the history of Dr. Buck's case, and states that the case had not previously been published. Comparing Mr. Poland's account of the case which he saw with the particulars of Dr. Buck's case, we notice some discrepancies, but these are readily explicable when it is remembered that Mr. Poland had lost his detailed account, and wrote from "scanty notes" eked out by memory. That Mr. Poland's case was not Dr. Van Buren's case is quite clear. Dr. Noyes was assured by Dr. Van Buren that his patient's *left* carotid was tied for aneurism in the *left* orbit, whilst Mr. Poland's patient was affected on the *right* side, and had a scar over his *right* carotid. There is, therefore, no room for Mr. Poland's suggestion that Dr. Van Buren wrote *left* instead of *right*. Mr. Poland states that his patient told him he had been treated by Dr. Mott; but this is certainly either a mistake of the patient or an error arising out of the loss of Mr. Poland's manuscript. To the same accident, and to the fact that Mr. Poland had read and transcribed Dr. Van Buren's case, we may fairly attribute the coincidence in the two accounts. If Dr. Buck's case was the case seen by Mr. Poland (and there seems to be no doubt of the matter), the patient must have attended at Moorfields in the latter half of 1858, or early in January, 1859. Mr. Poland gives no date, but his article was received by the editor in February, 1860 (see 'Royal London Ophthalmic Hospital Reports,' 1859–60, vol. ii, p. 219).

carotid artery successfully at the beginning of 1858. Both eyes were affected, but severer symptoms existed on the left side. In the same year M. Carron du Villards met with a case which was not submitted to treatment; Dr. Scaramuzza cured a patient affected idiopathically by means of intermittent digital compression; Hirschfeld reported a traumatic case in which, after death, a *small hole obstructed by a clot was found in the internal carotid artery in the cavernous sinus*, and two cases of ligature proved fatal. In Mr. Bowman's case all the signs of "intra-orbital aneurism" were present, and, on post-mortem examination, the careful research of Mr. Hulke *failed in discovering any arterial lesion. The cavernous sinus, the circular, transverse, and petrosal sinuses, were filled with clot softening in the centre, and the enlarged ophthalmic vein was plugged at its opening into the cavernous sinus.*[1] In Mr. Nunneley's case, a woman sixty-five years of age, *the left internal carotid artery was found enlarged in the cavernous sinus and surrounded by clot. The ophthalmic artery and its branches were enlarged.* In 1859 Mr. Corner, Surgeon to the Poplar Hospital, tied the right common carotid artery successfully in a traumatic case. The bruit persisted for twelve years and then ceased. Mr. Nunneley tied the carotid for the fourth time in an idiopathic case. Five years later the patient dying with symptoms of apoplexy, Mr. Nunneley found *an old aneurism of the ophthalmic artery at its origin from the internal carotid.* Previously to the occurrence of this case Mr. Nunneley had communicated a paper to this Society on "Aneurisms of or within the Orbit," in which he related his first three cases of the

[1] Mr. Hulke in his report of the case says that the majority of cases on record were not aneurism by anastomosis. There were two distinct series of cases. Of twenty-one cases known to Mr. Hulke the largest set consisted of true or diffused aneurisms, as shown by the suddenness of the attack, often after violence, &c.; the smaller set were aneurisms by anastomosis or erectile tumours. Mr. Bowman's case belonged to neither category ('Royal London Ophth. Hosp. Rep.,' April, 1859). Dr. Delens suggests that Mr. Hulke may have overlooked a small wound of the internal carotid, but Mr. Hulke assured Mr. Holmes that this could not have been the case (see Mr. T. Holmes's "Lectures on the Surgical Treatment of Aneurism," 'Lancet,' 1873).

affection. Having contended strongly against the belief that the cases previously recorded as such were really cases of "aneurism by anastomosis," he expressed his opinion that "some, if not the majority, were false circumscribed or diffused aneurism resulting from rupture of the vessel, rather than from dilatation of its diseased coats;" that in some the ophthalmic artery in the orbit was the seat of the disease; and that in some, as in his own third case, the carotid artery at its bend within the cranium was affected. Mr. Nunneley appears to have been unacquainted with the observations of Baron, Gendrin, Nélaton, and Hirschfeld, but his views indicate a decided advance in pathology, and had some influence in limiting the area of the doctrine of "aneurism by anastomosis."[1]

This doctrine was also combated ably and strongly by M. Demarquay, in a series of articles on intra-orbital aneurisms published in the 'Gazette Hebdomadaire for 1859,' and republished the following year in his "Traité des Tumeurs de l'Orbite." M. Demarquay divided the cases of intra-orbital aneurism into two categories, the *traumatic* and the *idiopathic*. The traumatic cases he regarded as dependent upon a rupture of the ophthalmic artery followed by an effusion of blood and the formation of a diffuse aneurism which he called a *primitive diffuse aneurism;* whilst, in the idiopathic cases, he supposed that there existed first of all a circumscribed aneurism of the ophthalmic artery, which was suddenly ruptured and became diffuse at the time of the commencement of the symptoms, and this form of aneurism he called *consecutive diffuse aneurism*.[2]

[1] Mr. Nunneley referred to the cases of Travers, Dalrymple, Busk, Scott, Curling, H. Walton, Velpeau, and Jobert (these two cases being communicated by Mr. Erichsen), Guthrie, and Van Buren; also to cases of aneurism by anastomosis treated by ligature of the carotid by Dr. Wood and Dr. Valentine Mott (see Dr. Wood's article in the 'New York Journal of Medicine,' 1857), and to Dupuytren's case of mixed erectile and cancerous growth, Schmidt's case of erectile tumour, and Freer's case of fungus hæmatoides. He omits all mention of the cases of Roux, Warren, Baron, Gendrin, Brainard, France, Critchett, Nélaton, Hirschfeld, Bourguet, and Gioppi—all of which had been published before his paper appeared.

[2] The evidence brought forward by M. Demarquay in support of his hypo-

Mr. Syme's successful case of ligature occurred in 1860, and was reported at the time by Mr. Joseph Bell, who recognised the distinction between the idiopathic and traumatic cases, and in an able article strongly opposed the doctrine of aneurism by anastomosis.[1] In the same year

thesis was of an inconclusive character. He cited the observations of Guthrie, Carron du Villards, and Giraudet of Tours. The cases of Travers, Dalrymple, Freer, Dudley, Roux, Jobert, and Herpin, he adduced as instances of *consecutive diffuse aneurisms*, and the cases of Busk, Scott, Curling, Velpeau, Petrequin, Brainard, Bourguet, and Gioppi, he ranged under the head of *primitive diffuse aneurisms*. M. Demarquay was more successful in his arguments against the doctrine of aneurism by anastomosis, and he justly referred to the entire absence of necroscopic proofs of the existence of aneurism by anastomosis in the orbit in any single case of intra-orbital aneurism. The weakness of his own theory in the same respect did not escape him, and he therefore brought it forward rather tentatively than positively, but he was strongly convinced of its correctness, and was able to adduce the pathological observations of Guthrie and Carrons du Villards as proving the occurrence of ordinary aneurisms in the orbit idiopathically, and the views of Busk, Curling, Petrequin, Bourguet, and Gioppi, who had all described their traumatic cases as instances of aneurism of the ophthalmic artery. He was also able to quote Mr. Hulke's opinions, and through Mr. Hulke those of Mr. Nunneley, as opposed to the doctrine of aneurism by anastomosis accounting for the majority of the cases of intra-orbital aneurism. Through a singular error, as already mentioned, Demarquay affirmed that the rupture of the ophthalmic artery diagnosed in Busk's case had been verified at a later date by an autopsy; whereas Busk's patient was alive and well when he last came under observation. Gendrin's case was brought forward as a case of phlegmasia of the orbital arteries. The existence of erectile tumours was not absolutely denied, and the cases of Rosas and Haynes Walton were adduced as probable examples of arterial erectile tumours of the orbit. I find neither mention of the cases of Baron Hirschfeld, Nélaton, France, Critchett, Scarramuzza, Van Buren, and Mott, nor allusion to the subject of arterio-venous communications.

[1] In illustration of the pathology of the affection, Mr. Bell referred to the post-mortem examination in Mr. Nunneley's fatal case of ligature, and to the very brief account of Mr. Bowman's case in the 'Lancet' of August 11th, 1860. He thought that an aneurismal condition of the internal carotid might account for the symptoms in some of the cases referring to Nunneley's fatal case, to Dudley's case of endocranial aneurism, and to a case reported by Sir Gilbert Blane, in which dilatations of the internal carotids much smaller than in Nunneley's case produced the most violent symptoms—acute pain, noise, and blindness. Aneurism, true or false, within the orbit he considered to be the cause in Scott's and Busk's cases. In Scott's case he says "there was evidently

Mr. Bowman tied the carotid with partial success for a pulsating tumour of the left orbit after the failure of digital compression. M. Passavant met with a case which followed an injury with a knitting-needle, and which he regarded as an aneurism of the lachrymal artery. The patient was a girl nine years of age. An attempt was made to reach the presumed aneurism by resection of a part of the external wall of the orbit, but it proved unsuccessful, and ultimately the child is stated to have remained in the same state as before. In 1861 Dr. Clarkson Freeman cured an idiopathic case by means of digitalis, cold, and direct mechanical compression of the pulsating tumour;[1] and Mr. Ernest Hart ligatured the carotid artery successfully in a case which he described in the following year as "a case of arterio-venous aneurism of the frontal branch of the ophthalmic artery and its satellite vein." Mr. Hart's able and comprehensive paper in the 'Lancet,' including references to most of the previously recorded cases, rendered the treatment by galvano-puncture, injection of coagulating fluids, and digital compression, better known by discussing the cases of Bourguet and Brainard, and furnishing an abstract of the cases of Gioppi and Scarramuzza.[2]

either a false aneurism or a rupture of an artery in the orbit;" whilst in Busk's case "the small aneurism could be felt." He then adduces Guthrie's case and two cases of aneurism of the arteria centralis retinæ cited by Mackenzie—one with pulsation in the orbit besides loss of sight." Lastly, he says that "obstruction to venous return in the sphenoidal fissure by endocranial aneurism or other tumour may be allowed as a cause, at least in cases where no true aneurism could be found on dissection, and especially in those cases occurring after blows on the head, and in connection with other head symptoms denoting internal mischief." Mr. Bell does not mention the cases of arterio-venous communication (see 'Edin. Med. Journ.,' 1861, p. 1064).

[1] One or two writers have stated erroneously that Dr. Clarkson Freeman's case was treated and cured by *digital* compression of the *carotid, e.g.,* Dr. Morton and Mr. Soelberg Wells.

[2] Mr. Hart gave a table of cases of ligature of the carotid for intra-orbital aneurism, 20 in number, of which 3 died, 1 was unsuccessful, 2 partially successful, and 14 successful. His table does not include the cases of Warren, Dudley, Haynes Walton, and Critchett, and he omitted the case assigned by Mr. Poland to Dr. Mott, probably regarding it with Mr. Poland as identical with Dr. Van Buren's case, which he included in his table.

Then followed Dr. Greig's successful case of ligature of the common carotid, and M. Legouest's successful case of ligature of both the common and external carotid after the failure of digital compression. Dr. Holmes, of Chicago, cured a traumatic case by the exhibition of veratrum viride and extract of ergot. Mr. Nunneley tied the carotid for the fifth time in a traumatic case, effecting a cure, and shortly afterwards for the sixth time with temporary relief in a doubtful case, which proved to be a case of cancer. A seventh case was seen, but the patient declined to submit to operative measures. In 1864 Mr. Nunneley's second paper, entitled, "On Vascular Protrusion of the Eyeball," was read at this Society. It contained his three last cases, and the account of the post-mortem examination of one of his earlier cases, at which he had found a circumscribed aneurism of the ophthalmic artery within the cranium. The views which he advocated may be gathered from the following passage from his paper:—"In the great majority of such cases of protrusion of the eyeball there is no disease whatever within the orbit: the seat of it is most commonly intracranial. The protrusion of the eyeball is passive, and the other distressing symptoms are secondary, depending upon obstruction to the return of blood through the ophthalmic vein. That this pressure in the great majority of acute spontaneous cases is caused by an aneurism of the carotid as it emerges into the cranium, or of the ophthalmic artery near its origin, is, I think, now certainly proved. In cases of traumatic origin supervening soon after injury to the head, the cause is most probably effused blood near to or within the cavernous sinus, whilst in those cases in which the protrusion does not come on until some time after the injury, or where the violence has not been so considerable, it may be serum or fibrine, or even pus, the result of suppuration within the sinus." Although Mr. Nunneley's pathological observations were not altogether complete, and although he was seemingly unacquainted with the published examples of arterio-venous communications in the cavernous sinus, he is entitled to the main credit of directing attention away from the orbit to the

vessels immediately behind it, and of throwing effectual discredit on the doctrine of "aneurism by anastomosis." In the following year Dr. Morton, of Philadelphia, reported an idiopathic case, cured by ligature of the carotid, as an "aneurism of the ophthalmic artery;" and, contesting the views of Nunneley, laboured to prove that the generality of cases of intra-orbital aneurism were ordinary aneurisms belonging to the ophthalmic artery. In a second paper published in 1870, Dr. Morton has modified his former views, and taken a more comprehensive view of the subject, and he has given accounts of several other cases which had fallen under his observation.[1]

In the same year occurred a case seen by Mr. Erichsen which subsided spontaneously under simple treatment; Szokalski's doubtful case ligatured with partial benefit, and M. Nélaton's second traumatic case. M. Nélaton tied the carotid, but the patient, a girl of seventeen, succumbed on the twelfth day. *A small hole was found in the left carotid artery in the cavernous sinus due to perforation by a detached fragment of bone.* The case related in abstract by M. Legouest was first published at length in 1870 in Dr. Delens very able and exhaustive monograph on communications between the carotid artery and the cavernous sinus; a work which has already exercised considerable influence in modifying current views on the pathology of intraorbital aneurism. In 1866, M. Desormeaux cured a traumatic case by injection of perchloride of iron, and a case under the care of M. Collard subsided under sedative treatment, whilst the patient was

[1] In his first paper Dr. Morton gave a list of thirty-four cases including Mr. Coe's case of intra-cranial aneurism and a case of vascular tumour of the orbit under Mr. Haynes Walton treated successfully by injection, and two cases of aneurism by anastomosis, one under Dr. Wood and the other under Dr. Mott (see 'Amer. Journ. of Med. Sciences,' 1865, vol. xlix). In his second paper he divided the cases of "vascular exophthalmos" into four groups: 1. True aneurisms of the orbit. 2. Diffuse aneurisms of the orbit. 3. Aneurisms by anastomosis. 4. Obstructed venous circulation intra- or post-orbital; and he enumerated what he considered to be their differential characters ('Amer. Journ. of Med. Sciences,' July, 1870, "Orbital Aneurismal Disease and Protrusion of the Eyeball from Venous Obstruction," &c.).

doing light work. Dr. Œttingen, of Dorpat, met with an idiopathic case which he attributed to thrombosis of the orbital veins, and he endeavoured to prove that blocking of the orbital veins, notwithstanding the anastomoses with facial and temporal veins insisted on by Sesemann, would occasion pulsation and bruit.[1] A post-mortem two years later disclosed an *absence of arterial lesion and partial obliteration of the orbital veins.* In 1867 Mr. Zachariah Laurence and Mr. Joseph Bell each tied the carotid successfully in a traumatic case. Mr. Laurence mentions another case under his observation, but I am not aware that it has been recorded. Dr. Foote met with a severe case following a fracture of the skull. The affection existed on the left side. Dr. Foote tied the left carotid, but as the symptoms were not relieved thirty days afterwards, he tied the right carotid. A month later the patient was discharged cured. In 1868 M. Socin tied the carotid in a traumatic case under the care of M. Schiess Gemuseus. At the end of five months the state of matters was much the same as before the operation. Pressure on the right carotid stopped the pulsation and bruit, but no further treatment was adopted. In the same year another fatal case of ligature occurred. The patient, a woman of sixty-three, under the care M. Wecker, was operated on by M. Richet, and died on the second day. *The ophthalmic vein was found very much dilated, and there was very marked atheromatous induration of the inner coat of the carotid artery.* Dr. Zehender published a summary of the results of ligation of the carotid for pulsating tumours of the orbit, and this was abstracted in the following year by Dr. Noyes, of New York. Dr. Zehender gave thirty-one cases: Dr. Noyes added others, and made up a total of

[1] Dr. Sesemann's account of the orbital veins is very different from that ordinarily given. According to him the ophthalmic vein sends a very small quantity of blood into the cavernous sinus, the greater part of the orbital blood flowing out by the facial vein (Reichert's und Du Bois Reymond's 'Archiv,' Heft 2, 1869, and 'Klin. Monat.,' Aug., 1869). M. Sappey finds an absence of valves in the ophthalmic veins. For Dr. Œttingen's views see 'St. Petersburger Med. Zeitschrift,' bd. xi, 1866.

forty-five, including cases of cancer, intra-cranial aneurism, and aneurism by anastomosis in the neighbourhood of the orbit.[1] Dr. Harlan treated a traumatic case unsuccessfully by digital compression.

In 1869 M. Wecker met with a traumatic case which was benefited, but not cured, by compression. Mr. George Lawson reported a case which he had not considered it advisable to submit to treatment, and M. Galezowski cured, or nearly cured, a pulsating tumour of the left orbit (developed three years after an injury) by means of intermittent digital compression continued for several months. M. Galezowski's second case of intra-orbital aneurism, met with in 1871, exhibited the affection on both sides. The patient was sixty years of age, and was not submitted to treatment. In 1871, Dr. Schmid, of Odessa, tied the carotid with partial success. In 1873 the first case of ligature of the carotid for intra-orbital aneurism occurred in Germany. The patient was under the care of Dr. Hippel, and the artery was tied on the antiseptic plan by Professor Schönborn with considerable benefit.

M. Julliard, of Geneva, has related a case under the title of a diffuse aneurism of the orbit, due to atheromatous degeneration of the ophthalmic artery, and Dr. Nieden, of Buchom, has recently reported a traumatic case which was much relieved, and, when discharged from hospital, seemed to be on the high road to a perfect cure after ligature of the carotid artery performed according to Lister's method.[2]

[1] 'Klin. Monat.' f. Aug. vi, 1868, p. 114, and 'Noyes' New York Med. Journ.,' 1869. There is an analysis of seventeen cases in Schmidt's 'Jahrbücher,' Nov. 4, 1859. Also in "Die Verletzungen des Auges, von Zander und Geissler, Leipzig and Heidelberg, 1864," thirty-one cases are given. In 1868 M. Léon Lefort wrote on "Ligature of the Carotid for Vascular Tumours of the Orbit," 'Gaz. Hebd.,' 1868, p. 551. He does not give references.

[2] Dr. Nieden states that taking the statistics of Morton, Noyes, and Pilz, he obtains 113 cases of ligature of the carotid for pulsating tumours of the orbit, of which 79 were cured, 14 were not benefited, 7 obtained partial relief, and 13 proved fatal. Now, in 1865 Morton gave 30 cases of ligature, and in 1869 Noyes gave 45, including all Morton's cases and 15 others. In 1868 Pilz gave most of those given by Morton and Noyes, together with some others, such as McGill's, McClellan's,

Mr. Lansdown, of Bristol, has described a traumatic case of pulsating tumour of the orbit as a "varicose aneurism of the nasal artery and vein." It was completely cured by cutting through the tissues of the upper lid, exposing the globular pulsating tumour, and ligaturing the vessels connected with it.

The subject of "orbital aneurism" was discussed at length by Mr. Holmes in his very interesting "Lectures on the Surgical Treatment of Aneurism" delivered at the Royal College of Surgeons in 1873, and I may take this opportunity of expressing my obligation to him for some valuable suggestions and references, when my attention was first drawn to the subject. I am also much indebted to my colleagues, Mr. Waren Tay and Mr. Reeves, and to Dr. Buller and Mr. R. W. Parker, for assistance in collecting the abstracts of foreign cases. It may be as well to add that, from the list of cases enumerated above, I have purposely excluded cases of pulsating cancer of the orbit for which the carotid has been tied, as, for instance, the cases of Lenoir, Hamilton, Woodward, A. B. Mott, Van Buren, and Halstead; the cases of aneurism by anastomosis in the neighbourhood of the orbit treated by ligature by Dr. Wood and Dr. Mott, and Mr. Coe's case of ligature of the carotid for intra-cranial aneurism. The cases of Parrish and Rosas have also been omitted. Some of these will be referred to under the head of diagnosis, nor have I alluded to the case in which Mc Gill successfully tied both carotid arteries within the space of a month for tumours of both eyes, called "vascular tumours" by Pilz and "fungous tumours" by Erichsen, and obtained a subsidence of the swellings, because I have been unable to obtain the number of the 'New York Medical

and Cadwell's cases of "erectile" or "vascular tumours of the orbit." Critchett's case is placed under the head of ligature for hæmorrhage (erroneously, for the bleeding occurred after the operation). At the outside, when all the cases are taken, there would not be a larger number than 50. The statistics obtained from Pilz were communicated to Dr. Nieden in a letter, but it is evident that the total of 113 must have been obtained by adding Morton's 30 cases, Noyes' 38 from Pilz, together, and thus reckoning the great majority three

and Physical Journal' in which the case is recorded. The exact nature of the case is therefore unknown to me.

In reference to Ullman's case of double ligature it is stated, in Mr. Erichsen's table of ligature of both carotids ('Surgery,' vol. ii, p. 76), that the patient was twenty years of age; the disease, "erectile tumour of the orbit;" that the left carotid was tied in 1823, and the right a year afterwards; and that death took place from hæmorrhage on the third day after the second operation. In Pilz's table ('Langenbeck's Archiv,' vol. ix, 1868, p. 328), however, the disease is described as affecting the "left ear."

I have also omitted Cadwell's successful case of ligature of the carotid for what is described as "erectile tumour of the orbit." The patient was a woman of sixty years of age, the disease had commenced a year previously at the lower border of the right eye, and spread down to the side of the nose. It will be found in Norris and Pilz's tables, and is related by Cadwell in the 'Boston Medical and Surgical Journal' for 1840. To McClellan's case of erectile tumour of the orbit, also mentioned by Norris and Pilz as occurring in 1825, in a girl five years old, as being of four and a half years' duration, and as cured by ligature of the carotid, I have been unable to refer, because it is recorded in vol. v of the 'New York Medical and Physical Journal,' to which I have not had access.

The following is the history of my own case.

On the 8th of July, 1873, Wm. Clark, twenty-four, platelayer, was stooping down on the line, when the iron step of a rapidly moving engine struck the right side of his head and prostrated him. A few minutes afterwards he was taken to the London Hospital; he was quite conscious, and answered questions readily. There were two lacerations of the scalp on the right side of the head above the ear, and between the two a depression of the parietal bone could be felt, but no bare bone could be reached through either wound. At a later period when the swelling had subsided, the depression was found to extend forwards for three inches to the frontal bone. The other symptoms were bleeding from the nose, great swell-

ing of the right eye, and sub-conjunctival effusion on both sides.

On the 17th, ptosis and paralysis of the internal, superior, and inferior recti were observed on the left side, and it was thought that the left eye was rather more prominent than the right; both pupils were dilated, and acted well. On the 24th the left pupil did not act; it remained fixed midway between contraction and dilatation. During the next fortnight the patient improved, and the orbital muscles, with the exception of the levator palpebræ, regained their powers of movement. There was some congestion of the eyeball with a little chemosis. All these symptoms were attributed to effusion of blood near the seat of fracture, occasioning pressure on the nerves and obstruction to the venous circulation. There was no pulsation or tumour. At this time, as usual, I gave up the charge of my patients for a few weeks to my associated colleague, Mr. Reeves. According to the notes furnished to me by my house-surgeon, Mr. Alfred Kebbell, no fresh symptom was noticed for ten or twelve days. On the 23rd of August pulsation of the eyeball was observed, and on stethoscopic examination a bruit was distinctly heard over the temporal fossa. The patient also could hear a noise in the head like wind blowing—he had not heard it previously; it was increased in the recumbent position, and on closure of the ears. Vision with the left eye was impaired. Pressure on the left carotid lessened, but did not stop the bruit and noise in the head.

On September 3rd, Mr. Reeves instituted digital compression of the left common carotid. Six dressers were employed, each compressing with his thumb for ten minutes at a time. As the patient could not bear the pain of compression after the lapse of two hours and a half, ether was given, and its administration was kept up for three hours. At the end of this time it produced so much retching and vomiting, that it was left off, and compression was continued without it for the remaining period. The chief results of the compression were the production of hoarseness, and diminution of the bruit and noise in the head. On the 15th of September

digital compression of both carotids was tried for two hours, but caused too much discomfort to be continued longer. Digital compression of the left carotid artery only for seven hours was maintained without improvement. At the end of the month a consultation was called in reference to ligature of the common carotid, but it was considered inexpedient to perform the operation.

Early in October, when I resumed the charge of my wards, the following was the condition of the patient. There was exophthalmos to the extent of about a quarter of an inch, the eyeball being pressed forwards, downwards, and a little outwards. The conjunctiva presented the ramifications of dilated veins; its lower half formed a thick transverse pad resting on and concealing the everted lower lid. The iris acted less readily than that of the other eye; the cornea was clear; there was ptosis; the eyeball could be moved by the patient in all directions; the hollow beneath the orbital arch was obliterated, but I could not satisfy myself that pulsation could be felt by pressure in this region. There was no pulsation of the eyeball; it had only lasted for a few days. A loud continuous bruit so exaggerated during the arterial pulse as to seem at first intermittent could be heard all over the head; it was much diminished, but not abolished, by pressure on the carotid. The patient remained in the hospital till January, 1874, when he went to the convalescent home at Brighton. Digital compression tried for twelve hours successively in November, and intermittent instrumental compression, had produced no appreciable effect. The patient remained at Brighton for six weeks. On his return the exophthalmos was more marked, and the sight of the left eye so much impaired, that he thought he should lose it altogether. The space between the globe of the eye and the orbital arch was filled on the inner side with a pulsating, soft, compressible and thrilling tumour. Mingled with the bruit which retained its former character of continuity with reinforcements during the arterial pulse, a high plaintive whistling note could be heard at intervals. The sound is called by French authors the *bruit de piaulement* or *miaule-*

ment, from its likeness to the mewing of a cat. It was only audible in front over the eye and the orbital margin. When the pulsating tumour was compressed, the bruit heard over the orbit at once ceased. Ophthalmoscopic examination by Mr. Buller and Mr. Tay showed dilated retinal veins and prominence of the margin of the optic disk. At a later period Mr. Couper observed in addition pulsation of the retinal veins.

The foregoing characters led me to concur in the opinion expressed by Mr. Holmes, who had seen the patient more than once, that the nature of the aneurism was arterio-venous, and that there existed a communication between the carotid artery and the cavernous sinus. A further trial of compression was made in three ways. Direct pressure by means of a pad and bandage was applied to the pulsating swelling, but it increased the congestion of the conjunctiva. Intermittent compression of the left common carotid by means of a collar tourniquet constructed for the purpose was kept up for many weeks without exerting any beneficial influence. At the end of May digital compression was tried once more. The artery was compressed for four hours daily during sixteen days, by Messrs. Fisher, Rees, Heyman, Hughes, Rawes, and Clippingdale, under the constant supervision of Mr. Bowkett, the house-surgeon. At first it arrested the pulsation, but it was observed that pulsation returned in the tumour during the continuance of very firm and effective pressure. The bruit was lessened, but never ceased. Compression of the right carotid did not appear to have any effect. On the cessation of digital compression the patient was still kept at rest in bed, and was ordered tincture of veratrum viride. Beginning with five minims three times a day, the dose was gradually increased to fifteen minims four times a day. Sickness supervened after the increase of the dose, and the pulse fell from 65 to 48. Pulsation and prominence of the pulsating swelling were slightly diminished, but not sufficiently after nearly three weeks' trial to give a prospect of permanent benefit.

The patient was anxious for the cure of an affection which would interfere with his earning his living, partly by

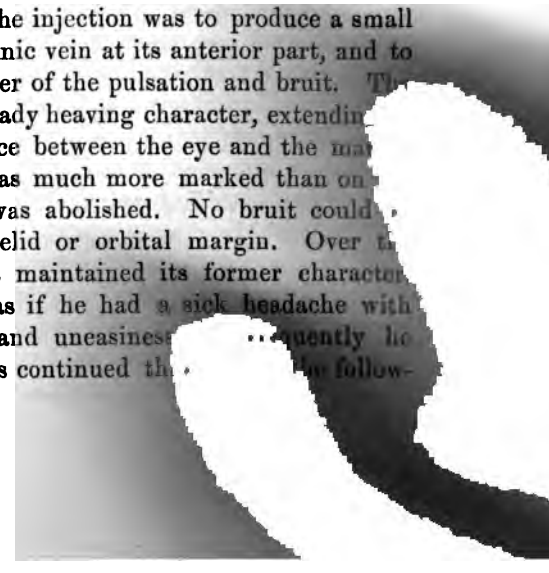
reason of its unsightliness, and partly because it was exacerbated whenever he undertook any exertion. A spontaneous subsidence was improbable. Removal of the eyeball was proposed by the patient, but it was explained to him that the operation would be likely to prove equally embarrassing and useless, and might necessitate ligature of the carotid. Galvano-puncture tried twice unsuccessfully, in other cases did not appear to be a suitable remedy for the form of aneurism. Ligature of the carotid would undoubtedly have been selected as the best method of treatment, but that the effect of digital compression seemed to show that the benefit to be expected from removal of the pressure of the blood in the left internal carotid artery would be but temporary, and that if the exophthalmos, pulsation and swelling were diminished or even removed for the time, they would in all probability return in a few weeks or months. The great desideratum appeared to me to be, to obtain the obliteration of the dilated ophthalmic vein. The agent best adapted for effecting this object seemed to be a solution of perchloride of iron. Injection of the perchloride had been successfully employed by Bourguet and Desormeaux, and strongly recommended by Broca, Demarquay, Delens, and others who had paid attention to the subject. On communicating my views to Mr. Holmes, he expressed the opinion that the information which we at present possessed was in favour of injection in preference to ligature, and in favour of further interference rather than of doing nothing. The comparative risks of the procedure by injection and ligature were explained to the patient, my impression being that injection would involve rather more risk to the eye, and be attended with some pain and discomfort, with sickness for a day or two, whilst ligature would entail more risk to life. The latter risk, however, I estimated at a low rate—the percentage of probable deaths being as I then thought about 15. The patient gave his consent to treatment by injection.

Accordingly on Saturday, July 11th, rather more than a year from the date of the accident which caused the aneu-

rism, I injected into the pulsating swelling below the orbital arch with a Pravaz syringe, five minims of a *neutral* watery solution of the perchloride of iron, of the strength of 28 per cent., in the presence of Mr. Holmes, who kindly assisted me, and my colleagues, Mr. Waren Tay, Mr. McCarthy, and Mr. Reeves. As at one of Mr. Nunneley's operations a violent thunderstorm was raging at the time. The patient was seated in a chair, and was not placed under the influence of an anæsthetic.

The pulsating tumour at the inner third of the orbital margin being very soft and repressible, and the tissues of the upper lid resisting, it was only at the second attempt that I succeeded in introducing into the venous sac the fine trocar and canula employed. This success was demonstrated by the issue of *arterial* blood through the canula on the withdrawal of the trocar. The syringe was then screwed on, and the solution injected. On withdrawing the canula no blood issued. Three or four minutes later it was observed that the upper eyelid was beginning to swell, and it gradually became tense, pressure on the carotid not appearing to exert any influence on it. The conjunctiva round the cornea was raised by clear transparent serum, which afterwards became turbid. Local pressure with a compress and bandage over the upper eyelid relieved the uneasy feelings of the patient.

Locally the effect of the injection was to produce a small coagulum in the ophthalmic vein at its anterior part, and to alter entirely the character of the pulsation and bruit. The pulsation became of a steady heaving character, extending the outer part of the space between the eye and the ma of the orbit. Here it was much more marked than on inner side. The thrill was abolished. No bruit could heard over the upper eyelid or orbital margin. Over t temporal fossæ the bruit maintained its former character The patient felt at first as if he had a sick headache with considerable local pain and uneasines ...quently he vomited, and the sickness continued th... ...w follow-ing day.



On Sunday the swelling had increased, and seemed to have reached its culminating point. The conjunctiva round the cornea was infiltrated and raised, so that the cornea was nearly concealed. The upper lid was distended, tense, and œdematous, and had the ecchymosed aspect which was observed in Bourguet's case, but this colour changed to a red inflammatory hue within twenty-four hours. The pulse was 48, the temperature normal. Effervescing ammonia mixture with a little hydrocyanic acid had been given to relieve the sickness, and hypodermics of morphia to relieve the pain. Ice-bags had been applied over the compress, but were too heavy for the patient to bear, the parts being very sensitive. On Tuesday evening, the third day after the injection, the swelling had increased, the cornea was concealed, the pulsation was stronger, the local discomfort was considerable, a slight epistaxis indicated an increase of pressure on the venous circulation. As a second injection would have been attended with difficulty on account of the distension of the upper lid, and could not be expected to lessen the swelling, I came to the conclusion that ligature of the carotid would be advisable, believing that it would relieve all uneasiness, and effect complete coagulation of the blood in the ophthalmic vein, favoured as it would be by the presence of pre-existing clot, that it would cause the subsidence of the infiltration round the eye by removing the vis à tergo, and that as the patient could still recognise objects held in front of the uncovered cornea, the operation would afford him the best prospect of recovering the use of his eye. Under these circumstances I determined to tie the carotid with as little delay as possible, if the measure should be approved at a consultation. Soon after 8 a.m. on the following morning, Wednesday, July 15th, Mr. Hutchinson, Mr. Tay, and Mr. Reeves saw the patient with me, and concurred in the proposal. The patient at first was unwilling to submit to the operation, but ultimately consented. He was placed under the influence of chloroform, as he would not take ether, and a silk ligature was applied to the left common carotid in the usual place above the omohyoid.

Owing to previous compressive treatment the fascia was found condensed and tough, and the tissues were so vascular that one or two superficial vessels had to be secured. The operation, performed as deliberately as possible, was unattended with any further difficulty than that resulting from the shape of the neck, and from the muscular development which was considerable, and rendered the vessel rather less accessible than usual. The jugular vein had a tendency to bulge over the artery inside the sheath, but being repressed with the finger gave no trouble. The sheath, being opened sufficiently to allow the passage of the aneurism needle, was gently separated from the artery on either side with a director, and the needle and ligature were passed.

On tightening the ligature, pulsation at once ceased in the upper eyelid and the tissues became flaccid. The bruit ceased for a minute, but was speedily reproduced, though much less loudly than before. No cerebral disturbance or other ill effects followed the operation. The ligature fell on the 5th of August, exactly three weeks afterwards. The swelling of the ocular tissues at once began to subside, and the arrest of pulsation was so complete that not the slightest beat or thrill could be felt in any part of them, and neither the frontal nor supra-orbital vessels could be detected. In a few days I was greatly disappointed at finding that, notwithstanding the steady removal of infiltration of the ocular tissues, a white spot which had appeared at the lower margin of the cornea had enlarged, and that loss of some portion of it was inevitable. A small ulcer formed, and some sloughing of the superficial layers occurred, whilst the rest of the cornea became hazy. As the upper eyelid was relieved it was ascertained that the ophthalmic vein contained firm clot, a hard rounded body occupying the place of the soft rounded pulsating swelling which had previously existed there. The patient went out in the middle of September. At that time I was away from the hospital, but he came to see me a month later. The eye had quite regained its position in the orbit, and the upper lid had to some extent recovered its power of movement. A narrow fold of

conjunctiva still projected, and the lower lid was everted. The cornea appeared semi-opaque and cicatricial, but the patient stated that he could distinguish light from darkness, and recognise his fingers moving in front of it. The bruit was not so loud as it was when he left the hospital.

On October the 24th I removed the projecting conjunctival fold with a pair of curved scissors, with a view to remedying the eversion of the lower lid. At the end of five or six weeks some eversion of the lower eyelid still remaining, strips of strapping were applied to bring and retain it in position. This treatment was effectual in a few weeks in removing the deformity. At the present time, therefore, the opacity of the cornea is the sole external indication of the original affection. At the latest date he could still distinguish light from darkness.

The main cause of the failure of the treatment by injection must, I think, be attributed to the small quantity of the solution of the perchloride employed. Being anxious to proceed with due caution, and following the recommendations of authors, I injected only five drops; ten or fifteen would probably have effected sufficient coagulation. The subsequent swelling was much greater than I had reason to expect from the records of other cases.

General Remarks.—The cases of intra-orbital aneurism fall naturally into two categories, those arising spontaneously and those following after an injury, or in other words the *idiopathic* and the *traumatic*. Of 73 cases referred to in the present paper, 32 were idiopathic and 41 traumatic. This classification is supported by a comparison of the two series of cases. Thus of 32 idiopathic cases 13 were affected on the left side, 15 on the right side, 2 on both sides, and in 2 the side is not stated. Of the traumatic, 41 in number, 27 were affected on the left side, 10 on the right side, 3 on both sides (in 2 certainly more severely on one side than the other), and in 1 the side is not stated. Hence it will be seen that the preponderance of cases in which the affection has existed on the left side, to which some authors call especial

attention, is entirely maintained by the traumatic cases, and must be regarded, I think, as of a casual character. This is shown also by the nature of the causes of the injuries occasioning the aneurisms. Of 13 cases due to blows of various kinds the disease occurred on the left side in 10. Falls occasioned the disease in 19 cases, 9 being on the right side, 8 on the left side. Punctured wounds in the orbit (2 with the ferrules of umbrellas, 1 with the forked end of the iron rib of a parasol, 1 from a fall on to a stick, and 1 from the entrance of a knitting needle), occasioned 5 cases. All the wounds were inflicted on the left side, but only 4 of the aneurisms occurred on the left side, the other being on the right side or side opposite to the seat of external injury. A discharge of shot received in the left orbit and eye occasioned the disease on the right side in 1 case.[1] One patient was wounded at the inner side of the left upper eyelid by the bursting of a soda-water bottle while he was in the act of stooping and opening a hamper. A pulsating tumour developed at the seat of injury. A blow on the nape caused the disease on both sides, but more severely on the right.

The average *age* of the idiopathic cases is higher than that of the traumatic. In the *idiopathic* cases the age ranged from four months and three weeks, in Mr. Haynes Walton's case (a case clearly of a special character), to 69, the mean age of 27 cases (Mr. Walton's case and one of Dr. Morton's being omitted) being just 43. In the traumatic cases the age ranged from 11 to 72, the mean age of 37 cases being between 30 and 31.

In regard to *sex* there is a further difference between the idiopathic and traumatic cases. Of 30 idiopathic cases, 21 were in females, and 9 in males. Of 41 traumatic cases, 31 were in males, and 10 in females.

[1] The case of Dr. Holmes, of Chicago. It is erroneously stated by Dr. Delens that the aneurismal symptoms occurred on the same side as the entry of the shot. But Dr. Holmes, who had seen Nélaton's first case, conjectured that the shot passed transversely through the tissues of the left orbit behind the upper part of the nose into the right orbit, wounding the ophthalmic artery or some of its branches.

The *mode of accession of the symptoms* deserves attention, and presents different characters in the two sets of cases. In the majority of the *idiopathic* cases the disease commenced suddenly with pain or noise in the head, or some peculiar feeling on the affected side; in other cases these precursory symptoms were absent. Two cases commenced in infancy without pain or constitutional disturbance, but both of these cases were different in kind from the cases occurring in adult life. Four cases occurred in young men and young women, to all appearance perfectly healthy. In two cases, both females, the affection was associated with disease of the heart and great vessels, and commenced suddenly on the left side with violent pains in the head and proptosis. In several there is no satisfactory account of the mode of origin. In a few the disease commenced gradually, with or without attacks of premonitory pain. Five cases occurred during pregnancy in women who had borne several children, four out of the five being six or seven months, and the other two months advanced in pregnancy. In all the attack occurred quite suddenly with violent pain and noise in the head, variously compared to a "sudden snap," the "cracking of a whip," the "report of a pistol overhead," a "blowing noise," and a "steam hammer," whilst in six other females not pregnant (three being over sixty years of age) in whom the attack was equally sudden, the several sensations experienced are described as "something appearing to give way," with or without a crack like that of a gun, "a kind of crowding feeling," a "strong buzzing," a "violent pain in the eye and temple," and "something queer in the head." Two of these patients were aroused from sleep by the pain and noise, in two the affection began as they were getting up as usual in the morning, and the others as they were pursuing some ordinary avocation, one being engaged in washing, another stooping down to take off her shoes, a third rubbing in a liniment ordered by her physician, a fourth returning from a drive, and a fifth sitting down at her work. One woman during an effort of childbirth felt "an unusual rattling in the eye," and perceived that "the eye was driven from the

orbit," whilst another attributed the affection to an effort made during her last confinement, five years previously. There is a strong resemblance between the whole of this group of cases pointing to a like resemblance in pathology. One man felt a deep-seated pain in the eye, and singing in the ear, as he was aroused from sleep. Another perceived one morning, after a fit of coughing, and without any precursory symptoms, that the eye was projecting from the orbit.

The first symptoms to appear after the precursory pains and noises have been generally exophthalmos and swelling of the lids and conjunctiva. Pulsation of the eyeball may be expected to precede the formation of distinct pulsating tumours in those cases in which both are observed. Bruit ought to be heard contemporaneously with the occurrence of the noises in the head, but as it is rarely listened for until pulsation is observed, the point remains to be determined. Exophthalmos has been extreme in a few cases, in two the eye is described as extruded from the orbit and lying on the cheek, in one even below the molar bone.

Of the *traumatic* cases it may be stated that the aneurismal symptoms are often masked by the general symptoms of injury to the head. As a general rule they are fully developed within nine months of the accident preceding the affection. More than half the cases exhibited all the symptoms within two months. Some of the cases were not seen till a much later period, but from the history it is clear that all the symptoms were well established a few weeks after the injury. In two cases only was there an interval of years.

Symptoms of fracture of the base of the skull are noted in 12 of the traumatic cases, in 17 the severity of the injury to the head renders it most probable that it occurred, whilst in 4 more it might not unfairly be presumed. Of the other cases 4 followed thrusts in the orbit, 1 was probably an idiopathic affection, 1 was occasioned by a discharge of shot, 1 by the explosion of a soda-water bottle and resulting wound in the upper eyelid, and 1 had several points of resemblance to a case of cancer, although it succeeded an injury. Setting

aside, therefore, 8 cases, we have 33 cases, of which 12 certainly, 17 almost certainly, and 4 probably had suffered from fracture of the base of the skull, more or less extensive and severe. This circumstance has an important bearing on the probable pathological cause of the affection in such cases.

The *primary symptoms* observed in the traumatic cases present some variations. In fourteen a blowing, roaring, or singing noise or beatings in the head are stated to have been heard either immediately or soon after the injury; in a few congestion of the conjunctiva and chemosis, with or without loss of power in the ocular muscles, and inaction of the pupil preceded exophthalmos, pulsation and bruit, so that in two or three cases the patients seemed to be affected with simple conjunctivitis. Exophthalmos is said to have preceded the pulsation and bruit in more than half the cases. It occurred directly or within a few days in 12, within a month or six weeks in 15, two months or a little longer in 2, five months in 2, and eight or nine months in 1.

The *development of a pulsating swelling beneath the upper eyelid at the inner third of the orbital margin* and *pulsation of the eyeball* are noted in both sets of cases. Of the idiopathic cases, 18 certainly presented a pulsating swelling between the eye and the orbital arch, and in 5 of these 16 cases pulsation of the eyeball was also observed. Of the *traumatic* cases, a pulsating tumour is noted in 24, pulsation of the eyeball also being specified in 8. Pulsation of the eyeball without a pulsating tumour *appears* to have been present in 6 idiopathic and 10 traumatic cases. In 7 cases pulsation is mentioned without distinct specification of the eyeball or of a pulsating tumour. Exophthalmos and bruit without either pulsation of the eyeball or a pulsating tumour appear to have been present in 3 idiopathic and 3 traumatic cases. The existence of bruit is specified in 22 idiopathic and 33 traumatic cases, but it was evidently present in all but two or three of an exceptional character. The exact characters of the bruit are noted only in a small minority. In a few cases the bruit was so loud as to be audible to bystanders.

The *typical* symptoms of the so-called "intra-orbital aneurism" are exophthalmos, a chemosed pad of conjunctiva concealing the lower lid, pain, paralysis of orbital muscles and iris, with or without anæsthesia, pulsation of the eyeball, a pulsating tumour above the eye beneath the inner part of the orbital arch, distension of conjunctival vessels, obliteration of the hollow beneath the orbital arch, bruit and noises in the head. Pulsation of the eyeball, a pulsating tumour, and paralysis of ocular muscles may be absent without contra-indicating the diagnosis of aneurism; but I do not think that any case should be regarded as aneurismal in which a bruit cannot be heard, or be placed under the head of "Intra-orbital Aneurism," unless, in the absence of pulsation, exophthalmos and congestion of conjunctiva accompany the bruit. In two idiopathic cases pulsation was apparently absent, and a bruit is not mentioned. One of these, a case of Mr. Nunneley's, was probably not an aneurism, for there were not even noises or beatings in the head; in the other, noises in the head were present, auscultation seemingly not having been practised.

The only true basis for the *pathology* of intra-orbital aneurism is the result of post-mortem examinations. Out of the 73 cases adduced in this paper, [illegible] opportunities for examination of the parts after death [illegible] recorded in 12 with the following results:

1. In one case only—the case of Guthrie—was found any aneurismal affection within the [illegible] case an *ordinary circumscribed aneurism* [illegible] *ophthalmic artery*. No account is given of [illegible] the blood-vessels behind the orbit.

2. In one case—the case of Dr. Œttingen—[illegible] inflammatory mischief [illegible] of the orbital veins was di[illegible] years later Dr. Œttingen [illegible] but evidences of past inflam[illegible] *partial obliteration of the* [illegible] the intra-cranial vessels is not [illegible]

3. In the remaining ten ca[illegible]

by some disease of the blood-vessels immediately behind the orbit.

4. In two of these ten cases *no arterial lesion whatever was discoverable.* One of the cases, Bowman's, was traumatic; and the other, Aubry's, was idiopathic. In both obstruction *to the passage of blood from the orbit through the intracranial sinuses* appeared to be the cause of the affection.

5. In one idiopathic case which Mr. Nunneley had cured by ligature, post-mortem examination five years afterwards showed a *circumscribed aneurism of the ophthalmic artery at its origin from the internal carotid.*

6. In three idiopathic cases—those of Baron and Gendrin and a case of Mr. Nunneley's—*rupture of an aneurismal internal carotid in the cavernous sinus* was discovered. In Mr. Nunneley's case the internal carotid artery contained and was surrounded by a clot. The ophthalmic vein and its branches would appear to have been very small, the opposite of the condition in Baron and Gendrin's cases, in which they were dilated. The ophthalmic artery and its branches were enlarged, and the inference from Mr. Nunneley's first description in his paper read at this Society undoubtedly is that these dilated branches of the ophthalmic artery had caused the pulsation observed during life. Further on in the same paper, the carotid artery is incidentally mentioned as "the ruptured and enlarged carotid."[1] From this description we should be quite unable to determine whether the condition causing the symptoms was rupture of an aneurismal carotid, simple dilatation of the carotid in the cavernous sinus, or aneurismal dilatation of the ophthalmic artery, and its two main branches within the orbit. In his subsequent descrip-

[1] 'Med.-Chir. Trans.,' vol. xiii, p. 186, line 19. At. p. 181 he says, "The ophthalmic artery was considerably dilated, its coats thickened with atheromatous patches; two of its branches, particularly the inner or the continuation of the trunk towards the inner angle of the orbit, were distended and filled with coagulum. The outer or lachrymal branch was also large and filled with coagulated blood, but not to the same extent as the inner. (It will be recollected before the operation it was at the inner side that the bruit and pulsation were most decided.) All the other branches, both of arteries and veins, were so small as hardly to be observed."

tion of the specimens, however, before the Pathological Society, he clearly speaks of the symptoms having been caused by a circumscribed aneurism of the internal carotid giving way in the cavernous sinus.[1]

7. In one case—that of MM. Wecker and Richet—the affection appears to have resulted from *simple dilatation and atheromatous degeneration of the internal carotid artery in the cavernous sinus*. It is not quite certain, however, whether the common carotid or the internal carotid in the cavernous sinus is the vessel indicated. No description is given of the state of the cavernous sinus and the sinuses connected with it. Hence it seems not improbable that the great dilatation of the ophthalmic vein and its frontal branch, which had formed the pulsatile thrilling tumours observed during life, may have resulted from the existence of a fissure of the internal carotid in the cavernous sinus which was overlooked at the post-mortem examination.[2]

[1] In the 'Transactions of the Pathological Society,' vol. xi, p. 8, Mr. Nunneley has given a description of the post-mortem appearances, together with a figure showing the left eyeball, the dilated ophthalmic artery, and the carotid surrounded by a clot. He heads the case: "A Circumscribed False Aneurism of the Cerebral Portion of the Left Internal Carotid Artery," and he speaks of the coagulum which surrounds the vessel as having escaped from the vessel. Further, in speaking of the pressure exercised on the ophthalmic vein and its branches, he observes:—"In the majority of cases this pressure is caused either by a small circumscribed aneurism of the internal carotid by the side of the sella turcica or of the ophthalmic artery, just at its origin, which may have given way, as in this instance, and as the history of Travers' and Dalrymple's cases would lead to the supposition had occurred in them (rather than as these two excellent surgeons imagined); or suppose the pressure results from effused blood escaping from a ruptured vessel in consequence of external violence, as in the traumatic cases in which Mr. Busk, Mr. Curling, and others in America and France have tied the carotid artery with success." From this it seems clear that in Mr. Nunneley's case an aneurismal dilatation of the internal carotid gave way in the cavernous sinus where the blood coagulated either before or after ligature, and, this being so, why were the ophthalmic vein and its branches smaller than usual? Could the ophthalmic vein by any possibility have been mistaken for the ophthalmic artery?

[2] After speaking of Bowman's case, Galezowski adds: "Another fact in all respects analogous is that of a patient operated on by Pro [illegible] by ligature of the carotid, from which the patient succumbed.

8. In three traumatic cases a *direct communication between the carotid artery and the cavernous sinus* was found. In one case the carotid artery was nearly torn across, in the two others there was a small hole in the artery.[1]

9. In no single instance has *aneurism by anastomosis* or *cirsoid aneurism within the orbit* been *verified by post-mortem discovery*. In none of the cases referred, to in which the diagnosis of aneurism by anastomosis was made, can an *undoubted* claim be established to that title. This diagnosis was strongly supported in the case of Mr. Haynes Walton, and has recently received the sanction of Mr. Holmes.[2] It seems to rest chiefly on the tender age of the patient and the arterial souffle heard with the stethoscope, and if correct an exception must be granted to the assertion that aneurism by anastomosis in the orbit would not be readily curable by ligature of a distant main artery. One of the two cases reported under the title by Dr. Morton (see Appendix, Case 28 in the 'Idiopathic Series'), presented the typical symptoms of arterio-venous communication, and pressure on the carotid controlled all bruit and thrill with immediate lessening of the orbital tumour; the other, if the tumour was prolonged into the orbit at all, exhibited very different features (Case 29 in the 'Idiopathic Series'). Some have regarded Warren's successful case as a case of aneurism by anastomosis, but I think without sufficient grounds. If it were really of this nature we should be able to adduce it as a fresh instance of the facility with which aneurism by anastomosis of the orbit may be cured by the ligature of a distant arterial trunk.

10. In no single instance has an *arterio-venous aneurism*

at the autopsy no aneurism was found, whilst the ophthalmic vein was greatly dilated" ('Maladies des Yeux,' t. i, p. 826).

[1] Mr. Bryant found the carotid artery divided in its passage through the petrous bone in a case of fracture of the base of the skull ('Practice of Surgery,' p. 49).

[2] "Aneurisms by anastomosis sometimes affect the vessels of the orbit, but the symptoms are quite different from those of the disease now in question." The only reference given is to Mr. Haynes Walton's case ("Lectures," 'Lancet,' Aug. 23rd, 1873).

been *found within the orbit at a post-mortem examination.* Mr. Hart diagnosed his case as arterio-venous aneurism of the frontal artery and vein, but he was not at that time acquainted with the cases of arterio-venous communication behind the orbit; and Mr. Holmes, who saw the case, considers the evidence decidedly in favour of a lesion of the internal carotid. Moreover, the subsidence of the exophthalmos and the removal of all trace of a pulsating tumour of the orbit after ligature, show that so far as the orbit was concerned the affection was cured, whilst the persistence of a loud whizzing bruit audible over the head is sufficient evidence of a morbid condition of a large vessel within the cranial cavity.[1]

11. In no case have the symptoms of intra-orbital aneurism been *proved* to be due to a *tearing across of the ophthalmic artery at or near the optic foramen.* It is clearly established, however, that the ophthalmic artery may be ruptured by violence in connection with fracture of the base of the skull. M. Demarquay adduces the instance of the unfortunate Dr. Bennati, who died in consequence of a fall upon the pavement. The eye was pushed forwards, and a fracture of the base of the skull with effusion of blood into the orbital cavity was diagnosed. At the autopsy it was found that the eye was pushed forwards by an enormous blood-clot, that a fracture of the orbit near the optic foramen existed, and that the ophthalmic artery and vein were torn across.[2] It is not improbable that the symptoms in Mr. Scott's case were due to a rupture of the ophthalmic artery consequent upon a fracture of the base of the skull. The occurrence of proptosis, and irrecoverable loss of vision directly after the accident, point to a tearing across of the ophthalmic artery and

[1] Concerning Mr. Lansdown's recent case I find it difficult to offer any positive opinion. The general distension of the orbital veins and exophthalmos would seem to contra-indicate a varicose aneurism of such small vessels as the nasal artery and vein, and the success of the treatment cannot be used as a conclusive argument in favour of the diagnosis. On the other hand, the absence of noises in the head and the fact that the bruit could not be heard over the head would certainly support Mr. Lansdown's views.

[2] Demarquay ('Traité des Tumeurs de l'Orbite,' 1860, p. 272).

the optic nerve close to the optic foramen. At the same time the symptoms are consistent with injury to the internal carotid, and the occurrence of violent arterial hæmorrhage from the nose decidedly favours this interpretation.

12. *Thrombosis of the cavernous sinus and ophthalmic vein* may be present without occasioning "intra-orbital aneurism." Exophthalmos, œdema of the orbit, hyperæmia, photophobia, dilated immovable pupil, paralysis of the muscles of the eye, and diminution of vision, are the evidences of this condition.[1] Hence I think that it is very probable that Mr. Nunneley's second case, in which the symptoms were œdema of the eyelids and chemosis with slight exophthalmos, little or no pulsation, no bruit or noise in the head, was not an aneurism at all, but a case of thrombosis of the cavernous sinus and ophthalmic vein.

13. *Aneurism of the internal carotid artery in the cavernous sinus* may be present without giving rise to the essential symptoms of intra-orbital aneurism. Mr. Holmes has related a case of a boy of sixteen, who died in St. George's Hospital from heart disease. At the post-mortem examination aneurism of the left internal carotid artery about the size of a small nut, filled with laminated coagulum, was found at the anterior part of the cavernous sinus. The third, fourth, and fifth nerves were stretched by the tumour. During life the orbital symptoms had been ptosis, dilated and fixed pupils, diplopia, impaired vision, and uncertainty in the movements of the eyeball.[2] [Mr. Hutchinson communicated

[1] See Knapp, 'Archiv für Ophthalm.,' 1868, i Ab., p. 207; also 'Annales d'Oculistique,' 1859-60, p. 186; Ogle, 'Brit. and For. Med.-Chir. Rev.,' 1865, "On Morbid Growths of the Brain, Spinal Cord, &c.," Case 281, p. 512; Cassou, 'Thèse de Paris,' 1857, "De la Phlébite de la Veine Ophthalmique;" 'Archiv. Gén. de Méd.,' Sept., 1874, "Ozène et otite purulente, &c., Phlébites des Sinus du Crâne et de la Veine Ophthalmique;" Dr. Œttingen, 'St. Petersburger Med. Zeitschrift,' Bd. xi, 1866.

[2] 'Path. Soc. Trans.,' 1861. M. Giraudet, of Tours, has reported a case of aneurism connected with the right internal carotid and ophthalmic arteries, obliterating the cavernous sinus and stretching the second, third, and fifth nerves, in a woman fifty-two years of age; but the observation throws no light on intra-orbital aneurism, because we are not in full possession of the orbital symptoms observed during life. The orbital symptoms mentioned are

to the Clinical Society, after this paper had been read, a most interesting case of a circumscribed sacculated aneurism

periodical pains in the head with a feeling of being struck by a hammer on the right orbit. This symptom had been present for ten years. There had been progressive loss of sight on the right side (see 'Gazette des Hôpitaux,' 5th March, 1857, p. 105; also Demarquay, op. cit., p. 294). Sir Gilbert Blane's case of aneurism of each internal carotid may also be referred to ('Transactions of a Society for the Improvement of Medical and Surgical Knowledge,' vol. ii, p. 193, 1800). In his 'Thèse des Tumeurs Aneurysmales des Artères du Cerveau,' Paris, 1866, M. Achille Gonguenheim cites the following cases of aneurism of the internal carotid: 1. M. Boudet's case of a dyer, aged fifty-seven, whose death was occasioned by the rupture of an aneurism of the right internal carotid a little before it gave off the anterior and middle cerebral. Previously to the fatal seizure he had been subject for two years and a half, on and off, to heaviness of the head, dimness of sight, and singing in the ear. 2. Dr. Moon's case ('Lond. Med. Gaz.,' 1848) of a woman of fifty-two, who died in consequence of a rupture of aneurism of the left internal carotid near its termination immediately beneath the optic nerve, which it flattened and elongated. The aneurismal sac had burst on the ventricular side, and the blood had penetrated through a fissure into the ventricles. There is no account of the earlier symptoms during life. 3. Holmes's case related by Dr. Ogle ('Brit. and For. Med.-Chir. Rev.,' 1865), with the age stated as 60 instead of 16. 4. A case under M. Bouley of a female, aged forty-three years, whose death was occasioned by the rupture of an aneurism of the left internal carotid and middle cerebral. She had experienced for some years violent pains, and she had been subject for some time to continual headache. 5. A case under M. Bigot of a lady, forty-six years of age, who became subject to violent pains, and one day felt great anguish in the head deeply and behind the left eye. The pain was subdued by treatment, but at the end of a year the left eye seemed to become smaller and retracted into the orbit with increase of pain and ptosis. Nothing could assuage her anguish. Six months later, after some improvement, she was seized with poignant pain in the head, uttered a piercing cry, and expired. The internal carotid artery was ruptured near the anterior clinoid process, the internal and middle coats having been destroyed and the external stretched and extended. My colleague, Mr. James Adams, reported in the 'Lancet,' December 4th, 1869, a case in which he found after death an aneurism of the right internal carotid artery, of the size of a walnut, projecting from the right cavernous sinus. The symptoms during life had been œdema of the lids and conjunctiva, hazy and ulcerated cornea, injected conjunctiva and sclerotic, and paralysis of the 3rd, 4th, 5th, and 6th nerves. For other cases see Breschet, 'Mémoires sur Differentes Espèces d'Anévrysmes;' a memoir, by Albers, of Bonn, 1836, on "Aneurisms of the Arteries of the Brain," Barth, 'Bulletins de Soc. Anat.,' 1849, p. 348; Dr. J. H. Hutchinson's paper, 'Pennsylvania Hospital Reports,' 1869; and Bartholow, in 'Amer. Journ. Med. Sci.,' 1872, vol. lxiv, p. 373, "On Aneurisms of Arteries at the Base of the Brain."

springing from the outer side of the left internal carotid artery in the cavernous sinus, and communicating with the carotid by a small aperture. The patient was a female, forty years of age. The tumour was as large as a pigeon's egg, and occupied the middle fossa of the skull. It appeared to have obliterated the cavernous sinus. There had been no orbital symptoms during life, except those due to pressure on the third, fourth, and fifth nerves ('Lancet,' 17th April, 1875).]

14. It is of the greatest importance to remember that in eight out of the twelve cases examined after death, the *ophthalmic vein and its branches* have been found varicose and enlarged. In four they had formed the pulsating swellings observed during life. This was the condition equally in Aubry's case of obliterated inferior petrosal sinus, in Wecker and Richet's obscure case, and in Nélaton's two cases of communication between the carotid artery and cavernous sinus. In Baron's case the varicose state of the orbital veins was the cause of the exophthalmos. In Bowman's and Gendrin's cases, the distended veins communicated their pulsation to the eyeball. In Guthrie's case the ophthalmic vein was enlarged, but no pulsating tumour could be felt. Pulsation of the eye probably existed, but is not mentioned. In the four remaining cases there is no record of dilatation of the ophthalmic vein. The typical symptoms of intra-orbital aneurism appear to have been absent in Hirschfeld's case, because the hole found in the carotid artery had been blocked up by a decolorised coagulum. In Nunneley's two cases little mention is made of the state of the veins. In Dr. Œttingen's case there are large gaps in the record. Hence there is no ground for inferring that dilatation of the veins did not exist during life in these cases.

15. Dilatation of the ophthalmic vein and its branches requires time for its development. In the early stages of the affection, therefore, a pulsating tumour may not be felt, but in progress of time it will appear, and the distension of the veins may extend to the forehead, the bridge of the nose, and the inner angle of the opposite orbit, and a furrow may be worn in the bone by the pulsating and dilated

ophthalmic vein. Before the dilated ophthalmic vein becomes evident at the margin of the orbit, its pulsations may be communicated to the eyeball, so that pulsation of the eyeball will often precede the development of a pulsating tumour. Ophthalmoscopic examination may be expected to display, in addition to changes in the disk and sometimes hæmorrhagic spots, engorged and perhaps pulsating retinal veins. The dilated and pulsating vessel or vessels observed on the forehead in a few of the cases, and regarded by one or two observers as arteries, must, undoubtedly, be viewed as dilated and pulsating veins.

16. When we endeavour to read the symptoms by the light of the various morbid conditions which have been found after death, the interpretation is easy in some cases and very difficult in others. We understand at once a circumscribed aneurism of the ophthalmic artery in the orbit giving rise to proptosis, pulsation of the eyeball, bruit, paralysis of orbital muscles, and enlargement of the ophthalmic vein. That admission of arterial blood from the carotid artery into the cavernous sinus either from wound or rupture of the carotid should cause dilatation of that sinus, gradual enlargement of the ophthalmic vein and its branches, the formation out of them of pulsating and thrilling tumours, soft, lobulated, and compressible, pulsating retinal veins, exophthalmos, bruit and noise in the head, and that it should be accompanied with various combinations of nerve lesion, we readily comprehend.[1] That in cases of arterio-venous communication, the opposite orbit and eye might become affected in like manner through the passage of arterial blood from the cavernous sinus on one side, through the circular sinus, into the opposite cavernous sinus and ophthalmic vein, is quite intelligible. We can also perceive that any obstruction

[1] According to M. Gendrin, the exophthalmia in his case was due to the dilatation and congestion of the ophthalmic veins and to the infiltrated hæmorrhage, which was produced around the cavernous sinus. The beatings were the result of the pulsation of the carotid, being transmitted by the infiltrated blood and the obliterated orbital arteries; the bruit was the result of lesion of the arterial walls. When the carotid became obstructed altogether the pulsation, &c. ceased ('Leçons sur les Maladies du Cœur,' t. i, p. 240).

to the return of venous blood from the orbit into the cavernous sinus, either from a dilated and atheromatous carotid or an aneurism of the carotid or intracranial portion of the ophthalmic artery, or from obstructed sinuses, might cause enlargement of the ophthalmic vein, congestion of the conjunctiva, chemosis, swelling of the eyelids, proptosis, and even the formation of a soft repressible tumour out of the enlarged ophthalmic vein, which should project between the globe of the eye and the orbital arch. We should expect, in cases of dilated atheromatous carotid, or aneurism of the carotid or ophthalmic artery within the cranial cavity, to find a bruit and to learn that the patient heard noises in the head. But we are at a loss to account for pulsation of the eyeball, or pulsation in the swelling formed by the enlarged ophthalmic vein. According to Mr. Holmes, the explanation offered by Mr. Nunneley of the pulsation observable in his cases of intracranial aneurism, was that the pulsation of the aneurism was communicated to the fluid in the enlarged veins, and so to the eyeball. But in his case of aneurism of the carotid he describes the ophthalmic artery and its inner and outer branches as enlarged, and the branches as filled and distended with coagulum, whilst all the other branches, both of arteries and veins, were so small as hardly to be observed. Moreover, he refers the greater distinctness of the bruit and pulsation at the inner side to the greater enlargement of the inner branch of the ophthalmic artery. This was in his first paper. In his second paper he explains the protrusion of the eyeball by obstruction to the return of venous blood by post-ocular pressure on the ophthalmic vein at its entrance into the cavernous sinus, but I cannot find anything more than an inferential explanation of the pulsation; and as Mr. Holmes has observed, "curiously enough, though Mr. Nunneley lays much stress upon the condition of the veins in explaining the pathology of the disease, he gives no distinct account of the state of the cavernous sinus, or of the venous system in connection with it, in either of his fatal cases."[1]

[1] "Lectures," 'Lancet,' Aug. 2nd, 1873, p. 143.

It is much to be regretted that the state of the internal carotid and of the cavernous sinus is not specified in M. Wecker's case, for as it at present stands we can neither account for the enormous enlargement of the ophthalmic vein, nor for the pulsation in it during life.

It appears to me that we must adopt one of two explanations of the pulsation observable in these obscure cases. Either pulsation is communicated from the carotid artery to the blood in the cavernous sinus, and so to the blood in the ophthalmic vein and its branches, or else it is transmitted from the arteries through the capillaries to the obstructed veins. The latter explanation would accord with the views of those physiologists who hold that pulsation may be transmitted to the blood in any vein in the body when the arterioles are much relaxed.[1]

[1] Dr. Flint says, "The intermittent action of the heart which pervades the whole arterial system is generally absorbed, as it were, in the passage of the blood through the capillaries; but when the arterioles of any part are very much relaxed the impulse of the central organ may extend to the veins. Bernard has shown this in the most striking manner in his well-known experiments on the circulation in the glands. When the glands are in physiological activity the quantity of blood which they receive is very much increased. It is then furnished to supply material for the secretion, and not exclusively for nutrition. If the vein be opened at such a time it is found that the blood has not lost its arterial character; that the quantity which escapes is much increased, and the flow is in an intermittent jet, as from a divided artery. This is due to the relaxed condition of the arterioles of the part, and the phenomenon thus observed is the true *venous* pulse. What thus occurs in a restricted portion of the circulatory system may take place in all the veins, though in a less marked degree. Except in the veins near the heart any pulsation which occurs is to be attributed to the force of the heart transmitted with unusual facility through the capillary system. A nearly uniform current is the rule, and a marked pulsation the rare exception. Mr. T. W. King, in an article on "The Safety-Valve of the Human Heart" ('Guy's Hospital Reports,' 1837), discussing the forces which concur to produce the venous circulation, mentions the fact that in some individuals, after a full meal, pulsation can be observed in the veins of the hand or the median veins of the forehead. This phenomenon is very delicate, and to make it more apparent he employed a thread of black sealing wax about two inches long, which was fixed across the vein of the back of the hand with a little tallow, so as to make a long and excessively light lever capable of indicating a very slight movement in the vessel. In this way he demonstrated

M. Aubry offers two solutions of the pulsation which was observed in his case in the ophthalmic vein. The first theory supported by M. Bérard was, that pulsation was communicated by the carotid to the blood in the dilated cavernous sinus, and thence to the veins in the orbit. The second theory, supported by M. Aubry himself, was that in consequence of the obstruction to the passage of the venous blood out of the cavernous into the inferior petrosal sinus, the capillaries became so much enlarged that they allowed the transmission of the force of the heart's action to the blood in the orbital veins, and this theory was considered by M. Aubry to be borne out by the circumstance that he was able to reproduce pulsation in the tumours by injecting the right carotid.

Concerning Mr. Bowman's case, Mr. Hulke has written forcibly as follows:—"It is difficult to explain the aneurismal symptoms by the pathological appearances, which were those of phlebitis of the cavernous, transverse, circular, and petrosal sinuses. The internal carotid artery may have been partially compressed by the swollen walls of the cavernous sinus against the side of the body of the sphenoid bone, giving rise to the bruit which would have a good conducting medium in the cranial bones. The plugging of the trunk of the ophthalmic vein where it joins the cavernous sinus, by obstructing the return of venous blood from the orbit, accounts for the protrusion of the eyeball, and perhaps also for the pulsation which was felt when the fingers were laid upon it; because each diastole of the ophthalmic artery must have been attended by a general momentary increase of the whole quantity of blood in the orbit, because its exit through the ophthalmic vein was cut off, and the resisting bony walls of the orbit could permit a distension in front only. The healthy state of the internal

pulsation in the veins of the hand, and also in the arm, foot, and leg. These movements are very slight, and are generally only appreciable by some such delicate means of investigation. In certain cases of disease Mr. King has noted very marked pulsation in the veins of the back of the hand and other vessels far removed from the heart" ('Physiology of Man,' vol. i, pp. 312-14).

carotid artery and its branches, and of the internal jugular vein, preclude the idea that the pathological changes in the cranial sinuses commenced subsequently to the deligation of the common carotid."[1]

Adopting these explanations offered by M. Aubry and Mr. Hulke, we may, I think, conclude that pulsation will occur in the orbit whenever the quantity of blood which enters by the arteries is to any considerable extent greater than that which the veins can immediately return. This will happen when the ophthalmic vein and its branches, or the cavernous sinus, is obstructed; the quantity of arterial blood which enters remaining either the same in amount as before, or being from any cause increased, whilst anastomosing venous channels do not suffice for the extra work thrown upon them. The former condition was that which existed in M. Aubry's and Mr. Bowman's cases; the latter would occur if the internal carotid artery in the cavernous sinus, and the ophthalmic arteries should be dilated from atheromatous degeneration, whilst obstructions from clot or other causes impeded or prevented the return current of venous blood. [But if this explanation be accepted, how are we to account for the absence of pulsation of the eyeball or pulsating veins in the orbit in such a case as that related by Mr. Hutchinson? Whilst we cannot agree with some anatomists that the cavernous sinus is not the main channel by which the venous blood passes out of the orbit, we must allow that a certain and even considerable amount of blood might be carried off by collateral channels. The special circumstances attending Mr. Hutchinson's case seem to afford the proper key. The origin of the disease was not, like that of most of the idiopathic cases of intraorbital aneurism, sudden or attended with any loud crack in the head, indicative of something having given way deeply in or behind the orbit. It was gradual, and the progress was slow. Moreover, the aneurism sprang from the outer side of the artery, and was a sacculated diverticulum from the main trunk; it was not an actual enlargement of the carotid

[1] 'Ophthalmic Hospital Reports,' 1859-60, vol. ii, p. 11.

in the cavernous sinus. As it increased the cavernous sinus was compressed, and in the end almost if not quite obliterated, but at the same time, I think, the trunk of the carotid was also compressed, and the ophthalmic artery which mounts up over the aneurism must have been compressed also. In this way, whilst the gradual progress of the disease allowed time for the establishment of collateral venous channels to relieve the ophthalmic vein and the sinus, the pressure on the carotid and ophthalmic arteries diminished the ordinary supply of arterial blood to the orbit. Lastly, there was no obstruction, as in other cases there may be, in the collateral channels themselves.]

17. In the traumatic cases there has generally been an interval of at least some days or weeks before the typical symptoms have been manifested. Assuming that the majority of the traumatic cases have been cases of arterio-venous communication in the cavernous sinus, I believe that the interval has been due to the blocking-up of the aperture in the artery by coagulum, this coagulum subsequently undergoing absorption or disintegration. In the early stage, therefore, the only symptoms have been some swelling and redness of the conjunctiva with slight proptosis due to obstruction of the venous current, but as soon as the aperture of communication has become clear, noises in the head, bruit, and pulsation of the eyeball have declared themselves. In my own case the patient named very precisely the date at which he first heard noises in the head, viz. about six weeks after his admission, and this date corresponded with the time at which pulsation of the eyeball and a bruit were detected. In some cases a partial rupture may occur and become complete after a variable interval, or weakening of the coats by injury may lay the foundation for an ordinary aneurism.

We pass now to the consideration of the *differential diagnosis* of the various morbid conditions of blood-vessels within and behind the orbit. Within the orbit we might meet with erectile tumours, venous or varicose tumours, pulsating encephaloid cancer, arterio-venous aneurism, ordi-

nary aneurism either circumscribed or diffused, and aneurism by anastomosis. Meningocele and derangement of the sympathetic system will also require a few words of comment.

1. *Erectile tumours,* which are of two kinds, *arterial* and *venous,* are not likely to be confounded with aneurisms, for they exhibit neither pulsation nor bruit.[1]

2. *Venous* or *varicose tumours* are formed by dilatations of the ophthalmic vein or its branches, owing to some obstruction to the return of venous blood. If formed by the ophthalmic vein they will be prominent at the upper and inner part of the orbit, and yield similar sensations to those afforded by the pulsating tumours found in cases of intra-orbital aneurism, in so far as this, that they will be peculiarly compressible and repressible, but will have

[1] It is to be understood that I use the term erectile tumour in a much more narrow sense than that adopted by Sir James Paget in his 'Lectures on Surgical Pathology,' and in an entirely different sense from Gosselin, Robin, Galezowski, and others. I do not either include or mean the cirsoid aneurisms or the aneurism by anastomosis, but simply tumours consisting either of capillary vessels or of a cellular, reticulated, and spongy tissue containing either arterial or venous blood, and often surrounded by a capsule of condensed connective tissue. Of such a character are the cases of cavernous tumour, four instances of which are referred to in works on ophthalmic diseases, namely, one recorded by Lebert, 'Abhandlungen aus dem Gebiete der praktischen Chirurgie,' Berlin, 1848, p. 88; one by De Ricci, 'Dublin Quarterly Journal,' 1865, Nov., p. 338; one by Von Graefe, 'A. f. O.,' vii, p. 12; and one by Wecker, 'Maladies des Yeux,' t. i, p. 798. In M. Lebert's case the tumour was congenital, and was removed by Dieffenbach. In De Ricci's case, a young woman of twenty-two, the tumour was removed, but a recurrence took place, and then Bowman removed it with the eye. In Von Graefe's case the eye and tumour were removed together. Wecker dissected out the tumour, preserving the eye. Cases of erectile tumours occurring to Abernethy, John Bell, Allan Burns, and Wardrop, will be found in Mackenzie 'On Diseases of the Eye,' 4th edition, 1854, p. 161, &c., or in the French translation, vol. i, pp. 227, 237—239.

Compare also Schmidt's case (vol. i, 'Ammon's Ophthalmological Journal,' 1831) referred to by Busk ('Med.-Chir. Trans.,' vol. xxii).

Dupuytren removed a tumour from the orbit, together with the eye, which was healthy, and found that it was composed of erectile tissue mixed with a certain small quantity of cancerous material (see 'Journ. Hebd.,' 1830, vol. vi, p. 75). Dr. Jeaffreson, of Newcastle-on-Tyne, has related a case of erectile tumour in

neither bruit, nor pulsation, nor thrill. They will increase in size, in expiration, and stooping, and diminish in inspiration and the erect posture.[1] Pressure on the internal jugular vein should cause them to swell.

the orbit. The patient was a lad of fourteen, pale and delicate. A slight protrusion had existed at birth, and since then had gradually increased. The symptoms were exophthalmos of the left eye to such an extent that the lids, which were rather œdematous, could not be closed over it, congestion of the ocular tunics, sluggish pupil, impairment of vision, absence of paralysis of ocular muscles, pain and tenderness, but occasional slight aching and feeling of tension. The floor of the orbit was occupied with a swelling which was deeply seated and had a hard semielastic feel, yielding on pressure or slipping into the orbit. Puncture did not give exit to fluid. On dividing the conjunctiva below the globe the tumour was found to extend deeply into the orbit and to surround the optic nerve. It was unconnected with the bones, from which it was easily separated, and the whole mass, together with the eyeball, was readily enucleated. There was very free hæmorrhage. The socket was plugged with lint dipped in a solution of perchloride of iron, and a rapid recovery took place. The tumour, which was somewhat lobulated and enclosed in an ill-defined capsule, appeared on section to be reticulated and spongy, and to be composed of a large quantity of blood-vessels connected by condensed cellular tissue ('Oph. Hosp. Rep.,' vol. vii, p. 187, 1873). Similar cases have occurred in the practice of Mr. Critchett and Mr. Hutchinson at the Ophthalmic Hospital. A good deal of bleeding sometimes occurs when the tumour is incised. For vascular tumours treated by injection of tannin by Mr. Haynes Walton and Mr. Taylor, see 'Lancet' or 'Med. Times and Gazette' for 1858. For the venous erectile tumours see Velpeau, art. "Orbite," Dict. en 30 vols., t. xxii, p. 310). Demarquay refers to three cases as related in abstract in the 'Annales d'Oculistique,' supplément iii, p. 44; one in the practice of M. Viguerie and the two others in the practice of M. Dieulafoy. They occurred in infants and their extirpation was followed by abundant hæmorrhage which was arrested by plugging.

Mr. George Lawson has reported an interesting case of nævus of the left orbit in a child, aged three years and two months. Six months previously the eye began to protrude, and was gradually pushed out of the orbit until the cornea suppurated. No tumour could be felt. The pain was constant and severe. Mr. Lawson excised the globe and removed the greater portion of the growth, which consisted of large veins with a cellular matrix. The child recovered without a bad symptom ('Lancet,' 1871, vol. i, p. 116).

[1] A remarkable case is recorded by M. Foucher in the 'Gazette des Hôpitaux,' 2nd Dec., 1858, and may be found in Dupont's thèse, "Tumeurs de l'orbite formées par du sang en communication avec la circulation veineuse intra-crânienne," p. 15. The patient came under the care of M. Nélaton, who cured the disease by two separate injections of perchloride of iron. M. Dupont relates two other

3. *Encephaloid cancer* when very vascular may be and has been mistaken for an aneurismal tumour, for it may possess both pulsation and bruit. Its onset is often

cases of venous tumour (Obs. 3 and Obs. 4, pp. 10—14). The first case was that of an infant sixteen days old, affected with a tumour between the globe of the left eye and the inferior eyelid, and having the aspect of a varix. During continued inspiration and crying it became distended, and the eye was pushed forwards. When the child remained quiet for seven or eight minutes the varix and exophthalmos both gradually disappeared. The reference given is J. A. Schmidt, cité par Himly, 'Ophthal. Biblioth.,' vol. iii, 1er cahier, p. 174, Jena, 1865. The second case was in a boy of 14, who had received the contents of a puff ball in his right eye at the inferior and external part three years previously. There was only pain at the time. Three months later the lad felt that the inferior eyelid swelled when he stooped. In the upright position there was no abnormal appearance, but when he lowered his head a tumour appeared of the size of an almond, soft, reducible, and without pulsation, and the skin became visibly distended and of a violet colour. The tumour gave the patient some inconvenience because he had frequently to stoop whilst working. He passed under the care of M. Serres, of Alais, who injected perchloride of iron into the tumour without success. (Obs. par Drs. A. Mazel et A. Bouiface d'Anduze, 'Gazette des Hôpitaux,' 23 Fevrier, 1861, p. 92. See also Demarquay, op. cit., p. 356.) The case of Dr. Parrish, related in the 'Amer. Jour. of Med. Sci.,' Oct., 1841, vol. i, p. 357, seems to have been of a similar character. A factory lad of 16 had a vascular tumour at the external angle of the right orbit under the upper eyelid with a prolongation under the skin of the lower lid. Enlarged and varicose veins were connected with it, and it seemed to derive its blood from deep-seated orbital vessels. When the eye was passive there was little to be seen. On everting the lids and making pressure on the blood-vessels of the neck the tumour became distended. It had originated after an injury six years previously received whilst diving. An attempt was made to remove the tumour by means of ligatures, but without success. In the article "Exophthalmia," in 'Cooper's Surgical Dictionary,' the following remarks are made by the author of the article:—"I remember a young lady who was referred to Mr. Laurence and myself by Mr. Maul, of Southampton, for advice respecting a tumour occupying the inner and upper portion of the orbit and attended with a degree of exophthalmia, constant exacerbation at the period of the menses, and occasionally double vision." The case of Rosas referred to by Gioppi was briefly this:—A scrofulous and dysmenorrhœal child received a violent contusion in the orbital region. Slight exophthalmos resulted. At the time of menstruation the eye became prominent, with thrill and profound pulsation. The affection was cured by bloodletting from the saphenous vein, leeches to the genital organs, and emmenagogues." This last case appears to be *sui generis*. Dr. Gruening's case of exophthalmos on lowering the head in 'Arch. of Ophth. and Otol.,' vol. iii, No. 1, p. 23, was probably a venous tumour.

obscure, and the case will be still more obscure if the orbital tumour exists alone, and if its development has followed an injury. Loss of vision, exophthalmos, pulsation, noise in the head, and bruit ceasing when the carotid of the corresponding side is compressed, are symptoms of cancer and symptoms of aneurism. Moreover the encephaloid tumour projecting outwardly may yield a soft semifluctuating sensation peculiarly liable to deceive even the experienced practitioner. In M. Lenoir's case the carotid artery was tied, under the impression that the affection was aneurismal. The disease had followed a fall on the back of the head, and the only symptoms of differential value were the light whispering character of the bruit, the irreducibility of the tumour, and its extension towards the temporal fossa.[1] Pulsation may be present without bruit, and the diagnosis will then be more obvious.

In Mr. Nunneley's case of cancer of the orbit the carotid artery was tied, and checked the progress of the disease; various opinions having been expressed in regard to the nature of the case. Its exact character was only ascertained at a late period of the disease. Here the diagnosis of cancer was rendered probable by the existence of multiple tumours, and by the continuation of the orbital tumour into the zygomatic fossa. The existence of a tumour in the left temple in Szokalski's case, continuous with the orbital tumour, the peculiar shape of the orbital tumour, and the subsequent appearance of a tumour on the left iliac crest, as well as the slight effects of ligature, are suggestive of cancer.[2]

[1] 'Bull. de la Soc. Chirurg.,' t. ii, pp. 61 and 84. Demarquay, op. cit., p. 473.

[2] Dr. Noyes gives the following cases of cancer as having been treated by ligature by American surgeons:—1854, Dr. A. B. Mott treated a malignant disease in the orbit successfully by removing the tumour and ligaturing the carotid; several previous extirpations had been followed by recurrence. In 1857 Dr. Van Buren tied the carotid for encephaloid cancer of the orbit; death took place on the thirteenth day from pyæmia. Dr. Woodward tied the carotid for a cancerous tumour of the orbit and brain. Death occurred in the eighth week. In 1858 Dr. Halstead tied the left common carotid of a girl of thirteen for a cancerous tumour which had been growing for three, years and a half; protruded from the outer canthus, pushed the eye forwards,

4. The distinctive features of *arterio-venous aneurism* within the orbit remain to be determined. [Mr. Lansdown's reported case is worthy of attentive consideration.]

5. According to Guthrie *ordinary circumscribed aneurism* within the orbit yields the typical symptoms of "intraorbital aneurism;" but his observation is so brief that it remains for subsequent observers to discover points of differential value. It is worth remembering, however, that no tumour could be felt by Guthrie within the orbit, and that in any similar case the bruit would most likely be of a distinctly intermittent character.

6. The special features of *diffused aneurism within the orbit* have still to be settled. Traumatic rupture of the ophthalmic artery would, in all likelihood, be accompanied by injury to the optic nerve and be followed by immediate protrusion of the eyeball, and immediate and irreparable loss of vision. The diagnosis of diffused intraorbital aneurism made in one or two cases does not rest at present on any solid foundation.[1]

and pulsated. The exophthalmos diminished, but soon increased more rapidly than before. The sight became *affected* in a few months. Nine months after ligature both the eye and the tumour were removed, and in two months the child was discharged cured. In 1860 Dr. Frank Hamilton met with a tumour in the right orbit in a child two years of age. It presented itself near the outer angle of the eye, was half the size of a Sicily orange, elastic, pulsating, and to the ear affording a rasping sound at each pulsation. The eye was pushed inward and protruding, and the sight of the eye was totally lost. The carotid was tied and caused cessation of the pulsation and bruit and diminution of the size of the swelling. The progress of the disease, however, was only temporarily delayed, and the child finally died of what proved to be a vascular malignant growth. In 1864 Dr. A. B. Mott successfully extirpated a cancerous tumour and tied the carotid. ('New York Med. Journ.,' 1869.)

[1] The term "aneurism by anastomosis," as Mr. Hulke has observed, has been used to comprise very various conditions. It was so used by John Bell, as may be seen by reference to his work on Surgery. But I think that the term is now and ought to be limited to cases in which there is a dilatation of distinct arteries anastomosing with each other, mixed or not with dilated capillaries, and not to tumours composed merely of a cellular structure or capillary vessels. In this sense I use the term in the text. This is the *arterial erectile tumour* of Paget 'Lectures on Surgical Pathology,' p. 581. It pulsates either "fully and softly" or "strongly," and has a distinct bruit. No doubt all the different forms of

7. If we should find reason to exclude Mr. Haynes Walton's case, neither *aneurism by anastomosis nor cirsoid aneurism* confined entirely to the orbit has been observed. In recorded cases which have been indisputably of this nature, and in which the orbit has been involved, the chief enlargement has been in neighbouring blood-vessels, and exophthalmos, congestion, and chemosis, &c., have been altogether absent, rendering the diagnosis easy. I cannot, however, agree with Nunneley, that "it is difficult to understand why Travers should have arrived at the opinion of his case being aneurism by anastomosis;" for although there may be some good grounds of distinction between cases of intraorbital aneurism and cases of aneurism by anastomosis and cirsoid aneurism, the resemblances between the two classes of cases in the aspect of the pulsating tumours and in the sensation imparted by tactile examination are far more obvious and striking than the points of divergence, and these latter are chiefly derived from the experience gained by the accumulation of observations since Travers wrote. What can be more natural than that a tumour formed by dilated and pulsating veins should be mistaken for one composed of dilated and pulsating arteries?

Compare the descriptions of the tumours in cases of intraorbital aneurism and the descriptions given by authors of aneurism by anastomosis, and the resemblance will be

vascular tumour run into each other, and it is difficult always to draw an absolute line of distinction, some cases appearing to be of a mixed character and to connect the classes of arterial, venous, capillary, and cavernous tumours together. All that I state in the text is that we have no *proof* of the occurrence of tumours in the orbit composed entirely or chiefly of *dilated arteries*.

On this subject see Robin, 'Gaz. Med.,' 1854; Gosselin's "Mém.," 'Arch. Gén. de Méd.,' 1867; Broca, 'Traité des Anévrysmes;' Laburthe, Thèse, 1867, 'Des Varices Artérielles et des Tumeurs Cirsoides.' Cases are included by these writers which, according to later views, were not cirsoid aneurisms at all, but instances of arterio-venous communications. Thus Laburthe relates the cases of Brainard, Bourguet, and Desormeaux; the first as an erectile tumour and the two latter as cirsoid aneurisms of the ophthalmic artery. Using the term "aneurism by anastomosis" in the widest sense, I quite admit that Mr. Walton's case is entitled to that designation.

at once obvious. The spongy feel, irregular shape, vibratory or purring thrill, compressibility, arrest of pulsation by pressure, either on the tumour or on the arteries leading to it, and the gradual extension of the disease are symptoms observable in both.

Intraorbital Aneurism.

Travers says, "The upper half of the inner canthus was filled by the thrilling tumour, which presented a loose woolly feel, was very compressible, and when firmly compressed offered a slight pulsation. The lower tumour, which projected above the suborbital hole, was of a conical shape and firmly elastic to the touch. It could be emptied or pressed back into the orbit, but the pulsation then became violent, and from the increased pressure of the globe the pain was insupportable. Careful compression of the temporal, angular, and maxillary arteries produced no effect on the aneurism. Upon applying my thumb to the trunk of the common carotid I found the pulsation cease altogether, and the whiz of the little swelling was rendered so exceedingly faint that it was difficult to determine whether it continued or not. The recent increase of puffiness in the skin over the root of the nose and below the inner angle of the opposite eye had given alarm to the patients and friends who feared, not without some appearance of reason, a similar affection of the right orbit."

Dalrymple says, "Deep seated between the integuments of the

Aneurism by Anastomosis.

John Bell says, "The tumour which I call aneurism by anastomosis is a perfect aneurism. It arises from some accidental cause; is marked by a perpetual throbbing; grows slowly but incontrollably, and is rather irritated than checked by compression. The tumour has only a sort of trembling or indistinct throbbing at first, but when it is fully formed it has a continual distinct pulsation; it beats strongly upon every occasional exertion; it swells up in spring and summer with a fuller and more active pulsation; it beats powerful in the time of menstruation; and by the incessant pulsation and occasional turgescence it forms among the cellular substance or among the dilated veins sacs of blood. These little sacs form apices and tender points, which become livid and very thin, and burst from time to time, and then, like other aneurisms, this aneurism pours out its blood so profusely as to reduce the patient to extreme weakness." ('The Principles of Surgery,' 1801, vol. i, p. 456.) Bell states that the disease affects all parts of the body, the eyelids included, and that it often begins in adults increasing from a tri-

eyelid a little towards the canthus of the eye, there was a cluster of small tumours of a firm and dense structure, causing great pain when compressed, and communicating to the finger a pulsatory thrill. Interposed between this cluster and the lower edge of the eyebrow, precisely in the course of the frontal branch of the ophthalmic artery, there was a hard tubercular substance which rose somewhat higher above the general surface of the eyelid, and pulsated still more distinctly than the smaller swellings. The texture of this substance was particularly hard and compact, the slightest pressure upon it occasioned intolerable pain. In addition to these appearances, immediately above the nasal third of the superciliary ridge, the integuments were gently elevated into a soft ill-defined tumour, occupying exactly the situation of certain branches of the frontal artery and pulsating simultaneously with the artery at the wrist. A similar elevation of the skin was perceptible at the root of the nose, giving a faint tremulous motion to a finger placed upon it."

M. Jobert says, "By the touch a tumour could be detected which had destroyed a part of the orbital arch; it increased rapidly and at the end of some months reached the frontal eminence. Pulsations synchronous with the pulse, expansile movements, and a species of susurrus like that no-vial pimple-like speck to a formidable disease. His first case was an aneurismal tumour about the size of a small egg, seated immediately over the eyebrow, and exactly in the course of the frontal artery. The tumour was covered with firm sound skin; its throbbing was exceedingly strong and had become very painful, &c. It is figured by Mr. Bell. The eye is not at all affected, but the question would naturally occur to Mr. Travers, what would be the effect of the disease if it occurred within the orbit?

Dr. Delens says, "Situated in the subcutaneous cellular tissue it is rounded, presents a notable relief, but at the periphery loses itself insensibly in the neighbouring soft parts. Its surface is lobulated and bossed, and yields to the touch the sensation of a bundle of worms. By touch, moreover, pulsations, synchronous with the arterial pulse, can be recognised. The tumour is readily reducible by pressure, but after this the finger often detects a furrow worn in the surface of the bone on which it rests. Auscultation reveals a bellows murmur, the characters of which vary according to the moment at which it is practised. Sometimes it is intermittent, sometimes it is continuous and jerky with redoublement," &c., &c. (Op. cit., p. 44.)

Mr. Erichsen says, "Aneurism by anastomosis forms tumours of varying magnitude and irregular

ticed in varicose aneurisms were perceived."

In Mr. Symes' case "the tumour felt not so much like an aneurismal sac as a bundle of small vessels; it was compressible and could be diminished in size, though not completely emptied. Pulsation was almost synchronous with the radial pulse, and accompanied by a sensible and audible thrill, very distinct at the orbital margin, the anterior edge of the roof of the orbit being in part, at least, absorbed."

The foregoing, to which others might be added, were idiopathic cases. In traumatic cases of long standing, the resemblance of the spreading and pulsating veins to the enlarged arteries of a cirsoid aneurism is very striking. Refer to such cases as those of Brainard, Bourguet, Desormeaux, and others, in which dilated and pulsating vessels could be seen on the forehead. That M. Desormeaux should have diagnosed cirsoid aneurism in his case can scarcely be said to be surprising.

shape; they are usually of a bluish colour, have a spongy feel, are readily compressible, not circumscribed, and have large tortuous vessels running into them on different sides. Their temperature is generally above that of neighbouring parts, and a vibratory or purring thrill, amounting in many cases to distinct pulsation, may be felt in them. This pulsation or thrill is synchronous with the heart's beat, may be arrested by compressing the tumour or the arteries leading to it, and returns with an expansive beat on the removal of the pressure. The bruit is often loud and harsh, but at other times of a soft and blowing character. These growths rarely occur in infancy, but generally make their appearance in young adults, though they may be met with at all periods of life, often as the consequence of injury." ('Science and Art of Surgery,' vol. i, p. 711.)

Some of the arguments which have been advanced by Busk, Nunneley, and others, against the view of aneurism by anastomosis, and the distinctions drawn between it and intraorbital aneurism, would appear to be not altogether valid. As distinguished from true aneurism and intraorbital aneurism, aneurism by anastomosis has been stated to be often of congenital origin, and to arise out of a nævus, to commence gradually and increase gradually, not to be caused by injury or violence, to involve neighbouring blood-vessels

which participate actively in the disease, to be confined to the cutaneous and subcutaneous tissues, to possess a feeble pulsation, especially when recent and small and a less audible bruit, and to be intractable to treatment by ligature of the main artery supplying it. Referring to Mr. Busk's arguments, Mackenzie, in his classical work on 'Diseases of the Eye,' states that the suddenness of the attack is the fact most indicative of true aneurism in the cases in question. Pulsation and distinct arterial susurrus attend aneurism by anastomosis. Rapid increase of the symptoms might attend true and anastomotic aneurism. Aneurism by anastomosis does not appear in every instance to arise from original malformation such as we observe in nævus maternus, but may begin in apparently healthy adults, from sudden and sometimes hidden causes. Neither is it confined to the skin or subcutaneous tissue, but affects indiscriminately all parts of the body, and brings on complicated morbid phenomena even among the viscera."[1] Again, in some cases cirsoid tumours have followed after injury, and the results of ligature—an argument *à posteriori*, and one which Mr. Busk says cannot be adduced to prove the nature of a tumour, are not quite so crucial as have been represented. Several cases of aneurism by anastomosis and nævus have been either cured or greatly relieved by the operation,[2] and in several cases of intraorbital

[1] This is taken from 'Bell's Surgery,' vol. i, p. 456. Bell refers to the cases of pulsating spleen related by old authors, and especially to the case described by Tulpius. "The patient was a poor labouring man, and Tulpius one day going to visit him along with Henricus Salius, a physician, they both heard the pulsation of his spleen at the distance of thirty feet, and that so distinctly that they could count the pulse as fairly by the ear as by the finger," p. 473.

[2] See 'New York Med. Journ.,' 1857, "Early History of Ligature of Carotid," for successful cases of Dr. Wood and Dr. Mott. In 1822 Pattison tied the carotid successfully for an "aneurism by anastomosis." A. Robert, Kuhl, and Warren, obtained cures by double ligature. Other surgeons have been less happy. Wardrop's cases of ligature of the carotid for nævus might also be cited. In Zeis' case of ligature of the carotid for nævus of the face in a child fifteen months old, although the patient died from convulsions on the forty-ninth day, the nævus disappeared. Rogers, Wilbaume, McClellan, and others more or less successful, are mentioned in Pilz's tables.

aneurism either only partial relief or no relief at all has resulted, or the symptoms have returned in a few months.

After all it may greatly be questioned whether argument alone would ever have abolished the doctrine of aneurism by anastomosis as applicable to the explanation of cases at least of idiopathic intraorbital aneurism. In the absence of pathological observations the tendency was to divide observers into two sets—those who adhered to the views of Travers and Dalrymple, and those who were convinced by Mr. Busk's arguments and the arguments of M. Demarquay, and adopted the opinion that the vessel implicated was the ophthalmic artery in the orbit.

It is to the records of post-mortem examinations that the disproof of the doctrine of aneurism by anastomosis [1] or cirsoid aneurism is due, and that we have been taught to look away from the orbit to the blood-vessels lying behind it for the origin of the affection.

8. A *meningocele* of small size may project at the internal angle of the orbit, and be mistaken for a nævus or erectile tumour. This happened in M. Guersant's case, which was seen by many Fellows of the Surgical Society of Paris, who agreed in recommending treatment by thread setons. Cerebral symptoms supervened, and the child soon died. It was found at the post-mortem examination that the tumour was composed of a small portion of brain substance covered by the membranes of the brain, which had passed through the fronto-ethmoidal suture, and appeared at the inner angle of the orbit.[2] *Spheno-orbital meningocele* is a disease of very rare occurrence, and its distinctive characters cannot be stated very positively. Dr. Œttingen, of Dorpat, has recently related an interesting case, in which he suspected that meningocele was associated with some arterial angiomatous tumour.

A lad of 14 had struck his head on a step when a year

[1] Mr. Holmes, in his lectures already quoted, observes, "Travers' original assumption that orbital aneurism is usually anastomotic has been conclusively refuted." ("Lectures," 'Lancet,' 1873, vol. ii, p. 255.)

[2] Guersant, 'Maladies des Enfants,' p. 246.

old. Subsequently during thirteen years his left eye had gradually become prominent. The upper eyelid was greatly swollen; the eyeball projected considerably; a tumour could be felt above and to the outer side of the eyeball. It pulsated, and could be emptied by pressure, but quickly filled again. Compression of the carotid checked the pulsation. There was no bruit to be heard. The left side of the head was curiously misshapen, and there was an occipital meningocele of small size having a very narrow channel of communication with the interior of the skull.

Langenbeck's plan of injection of ergotine and compression of the carotid were tried without avail. The left common carotid was ligatured. The tumour diminished in size and ceased to pulsate. Pulsation returned in four hours, but not so evidently. In a few days an attack of pneumonia supervened. The ligature fell in a fortnight, and the wound healed in a month. The tumour did not become more solid. Pressure on it made the occipital tumour swell out, and tapping the former with the finger produced a corresponding impulse in the latter; compression of both jugular veins made both tumours very tense. Compression of the right carotid checked pulsation, but when continued for a time had no curative effect. The ligature of the carotid was followed by considerable benefit.[1]

9. M. Collard supposed that in his case of intraorbital aneurism the accident occasioning it had given rise to a morbid state of the lenticular ganglion, perverting the action of the vaso-motor nerves and thus causing dilatation of the ophthalmic artery and its branches, and he is half supported by Mr. Erichsen, who asks, is it possible that some derangement of the vaso-motric influence of the sympathetic may really occasion the symptoms of increased vascular activity that are so characteristic of this singular disease? Having regard to the phenomena exhibited in Graves' disease, it would be extremely rash to say that the symptoms of intraorbital aneurism might not possibly be simulated in the

[1] 'Klin. Monat.,' Feb., May, 1874. Abstracted for 'Oph. Hosp. Reports, 1874.

same way as aneurism in the neck is simulated by the pulsating and thrilling thyroid gland. But it may be safely asserted that in none of the cases which have been referred to in this paper is any such explanation either demanded or permissible, whilst the fact that pulsation of the eyeball is never seen in Graves' disease seems to indicate that derangement of the sympathetic system is insufficient to occasion the symptoms of intraorbital aneurism. The case which forms the basis of Mr. Erichsen's query is that of Mr. Bowman, in reference to which Mr. Erichsen states that "no trace of aneurism or other *vascular* disease could be found on dissection." The words should have been "no trace of aneurism or other *arterial* disease could be found on dissection," for there was abundant derangement of the venous system, and the whole point of the case is that the symptoms of intraorbital aneurism may be occasioned by morbid states of the veins and sinuses—a conclusion which has no relation to the idea of a functional derangement of the orbital arteries.

The *differential diagnosis of the various conditions of the blood-vessels behind the orbit* which have been met with in cases of intraorbital aneurism still requires elucidation. So far as our information at present extends it would appear that—

10. The distinctive signs of *arterio-venous communication in the cavernous sinus* are a *bruit which is continuous with reinforcements during the arterial pulse,* and the *bruit de piaulement.* A vibratory *thrill* in the pulsating vein is regarded by surgical writers as characteristic of arterio-venous aneurism. It must be borne in mind, however, that thrill was absent in both of M. Nélaton's cases of perforated internal carotid, and that it was present in M. Aubry's case of obliterated petrosal sinus. Thrill was noticed in several traumatic cases which resembled M. Nélaton's in other respects, and in some of the idiopathic cases which were probably of a different nature. Moreover, in M. Aubry's case there was a bruit which was continued with reinforcements during the arterial pulse. On the

other hand, the *bruit de piaulement* was present in both M. Nélaton's cases, and may prove to be pathognomonic of communication between the carotid artery and cavernous sinus.[1]

11. In *traumatic* cases the previous occurrence of symptoms of fracture of the base of the skull, or the infliction of an injury to the orbit by a thrust from a pointed instrument, would afford strong presumptive evidence of arterio-venous communication. A careful consideration of the mode of occurrence of the injury may yield important information, as it did in M. Nélaton's first case.

12. In *idiopathic* cases advanced age and associated disease of the heart and great vessels would be strongly indicative of atheromatous degeneration and enlargement of the internal carotid in the cavernous sinus or of the ophthalmic artery.

13. In cases in which a pulsating tumour can be felt between the eye and orbital margin, the feel of the tumour would assist the diagnosis. An enlarged ophthalmic vein is at once revealed by its peculiar softness and compressibility as well as by its position near the inner angle of the orbit.

14. A differential diagnosis would certainly be much aided by puncturing the pulsating tumour above the eye with a small trocar and canula. If the blood which issued proved to be *arterial*, the existence of an arterio-venous communication might safely be predicted, provided that the other characteristic symptoms of that condition were present. If these symptoms were absent, and if the bruit were intermittent, the affection would probably be a circumscribed aneurism within the orbit.

If the blood issuing from the canula proved to be *venous*, the diagnosis would rest between obstructed sinuses

[1] The *bruit de piaulement* was present also in the case of Dr. Holmes, of Chicago, who observed it during the administration of veratrum and ergot, in the cases of Desormeaux, in Galezowski's unilateral case, in Dr. Nieden's case, and in my own case. Still, in Bowman's case of obstructed sinuses the bruit was loud and *sibilant*, and therefore much stress cannot be laid upon this peculiarity.

and an aneurismal affection of the carotid or ophthalmic artery causing obstruction to the return of venous blood.

It must, however, be recollected that puncture of the tumour may be attended with ill effects, and cannot be recommended for general adoption.[1] There would not be the same objection to puncture of a pulsating vein beyond the margin of the orbit.

15. Observations have been neither sufficiently numerous nor precise to determine beyond question the distinctions between *aneurisms of the carotid, aneurisms of the intracranial portion of the ophthalmic artery,* and *obstructed sinuses.* Still I think we might arrive at a correct conclusion by attention to the following particulars:

(*a*) *The sudden onset of the disease* with pain, and noise like the cracking of a whip or report of a pistol, and feeling of something having given way in the orbit, is indicative of the formation of an aneurism or of its rupture.

(*b*) The *presence of paralysis of the orbital nerves* is indicative of an aneurism, or of a ruptured aneurismal carotid surrounded by blood clot in the cavernous sinus pressing upon the nerves. The third nerve was implicated in Gendrin's case, in both Mr. Nunneley's cases, and in Wecker's fatal case. On the other hand, there was no nerve lesion in M. Aubry's case, and in Mr. Bowman's case the pupil was dilated but active, and neither ptosis nor loss of power in the other branches of the third nerve occurred till thirteen days after ligature, when probably the clots in the cavernous sinus or effusion into its walls increased and compressed the nerve.

(*c*) *Complete loss of vision at the outset and failure to recover it after ligature or digital compression* would favour

[1] In Brainard's case, Nunneley's first case, Bourguet's case, and my own (all traumatic), and in Jobert's idiopathic case, puncture of the pulsating tumour gave exit to *arterial* blood. Puncture exercised an injurious effect in three cases—that of Roux, in which venous blood issued, that of Nunneley, and that of Jobert. See Appendix. In M. Jobert's case acupuncture appears to have been injurious to vision.

the supposition of an aneurism of the ophthalmic artery rather than of the carotid.

(*d*) A distinctly *intermittent bruit* would point to true aneurism.

(*e*) A very *soft bruit continuous with reinforcements* and accompanied by thrill in the pulsating tumour, but unaccompanied by nerve lesions of any kind, would indicate a condition similar to that found in M. Aubry's case, and would contra-indicate a coagulated condition of the blood in the cavernous sinus.

16. *With reference to the nature of the recorded cases* I believe that nearly all the traumatic cases were examples of arterio-venous communication more or less free in the cavernous sinus. The evidence for this conclusion is strong. Three out of four fatal traumatic cases in which a post-mortem was obtained exhibited a wound of the carotid artery within the sinus. The pre-existence of severe injury to the head and in a large number incontestably of a fracture of the base of the skull, the similarity in the mode of origin, development, and nature of the symptoms, the pulsating swelling becoming evident at one spot beneath the upper eyelid in the situation of the ophthalmic vein, the peculiar feel of the tumours, the frequent extension of the pulsation to neighbouring vessels which without reasonable doubt have been veins, and the resemblances in the characters of the bruit all point in one direction. The occurrence of the symptoms on both sides in three cases is readily explained by the supposition of a free arterio-venous communication. I was very much struck with the strong resemblance between my own case and M. Nélaton's second case. In the idiopathic cases I am inclined to think that the most frequent condition has been the sudden formation or rupture of an aneurism of the internal carotid artery in the cavernous sinus. The intense pain at the back of the orbit and headache, the sudden snap or crack heard by the patient in a moment and sometimes without any precursory symptoms, the records of the post-mortem examination in the cases of Baron, Gendrin, and Nunneley, the analogy afforded by the symptoms and

necroscopic appearances in cases of ruptured aneurism of the internal carotid after it has left the sinus, the absence of the symptoms of "intra-orbital aneurism"—pulsation, exophthalmos, chemosis, &c., considered to be characteristic of obstruction to the return of venous blood from the orbit in the cases recorded by Holmes and Hutchinson, and in cases of obstructed sinuses related by Knapp and Ogle, seem to me to be strongly in favour of rupture. Aneurisms of the intra-cranial portion of the ophthalmic artery are of very infrequent occurrence. Aneurisms of the ophthalmic artery in the orbit have been found but once or twice, and we have no proof whatever of their rupture during life. Morbid conditions of the orbital veins and intra-cranial sinuses will probably prove to be exceptional causes of the group of symptoms which is covered by the term intra-orbital aneurism. In the present state of our knowledge it is impossible to dogmatise, and it remains for future observers to throw full light upon the pathology of the affection as well as to test the value of the suggestions which I have ventured to make in reference to diagnosis. Post-mortem examinations should be conducted with very great care, and where practicable, as suggested by Dr. Delens, it would be well to remove about the anterior half of the skull for deliberate dissection.

In regard to *treatment* the first question which arises is, what would be the probable course of the affection if left either quite alone or with only medical treatment? We have some data which may assist us in affording a reply. We have four traumatic cases cured by simple means—that of Mr. France in eight months; that of Mr. Erichsen in fourteen months by attention to habits of life and abstinence from stimulants; that of M. Collard in two years and a half by means of leeches, cold lotions, calomel, and belladonna, and light work; and that of Dr. Holmes, of Chicago after the exhibition of tincture of veratrum viride and Tilden's fluid extract of ergot for about six weeks. Then we have the idiopathic case of Dr. Clarkson Freeman cured by the application of cold, the exhibition of digitalis in small doses,

and mechanical compression of the pulsating tumour; the return of the symptoms on the opposite side in M. Herpin's case after ligature removed by the constant application of ice for three months, and the case of M. Julliard, of Geneva, subsiding, it may be, spontaneously, but in all probability assisted materially by the digitalis taken, and the ice applied for about a fortnight to the eye. The cases of Guthrie, Baron, Aubry, Hussey, Hirschfeld, and Carron du Villards, died without surgical interference; but we cannot say that the result was in any way influenced by the absence of surgical assistance. In M. Gendrin's case the eye retired into the orbit, and bruit and pulsation ceased whilst the patient was taking digitalis, but the cornea ulcerated and the anterior chamber became filled with pus, and in fourteen days the patient died. These cases stand on neutral ground. To oppose the expectant treatment, however, we have M. Nélaton's traumatic case dying from hæmorrhage from the nose, only mechanical compression of the carotid having been employed. We have Scott's case probably saved from a similar fate by prompt ligation of the carotid, and we must add those cases which had not improved or had actually deteriorated after a considerable interval of time. There was Travers' patient in whom the affection had existed for three years and five months without improvement; there was M. Jobert's patient who had been under a variety of practitioners for three years; there was Bourguet's traumatic case, which had been gradually getting worse for two years and six months; and there was Mr. Hart's traumatic case in which the symptoms had commenced three years previously to the time at which the patient first came under observation. Besides these cases there were the cases of Nunneley, Bell, Desormeaux, &c., in which at the end of a year the symptoms were aggravated.

The surgical means of treatment at our disposal are local compression of the tumour, digital and instrumental compression of the common carotid, galvano-puncture, subcutaneous injection of ergotine, injection of coagulating fluids, and ligature of the carotid artery or arteries.[1]

[1] To these methods ought to be added ligature of vessels within the orbit.

Local compression has been tried in 10 cases, 5 idiopathic, and 5 traumatic. It aided if it did not effect the cure in 1 idiopathic case (Clarkson Freeman's), produced some benefit in 2 traumatic cases, and was either useless, too painful, or injurious by increasing the chemosis and congestion in the remaining 7 cases.

Instrumental compression of the common carotid has been tried in 4 cases, all traumatic. One case died of hæmorrhage from the nose, and in the other 3 no benefit resulted.

Digital compression of the common carotid first practised by Gioppi has now been frequently employed. Of 16 cases in which it has been applied, 5 were idiopathic, and 11 traumatic. Of the 5 idiopathic cases 2 were cured, the bruit and noise in the head persisting in 1, and vision, nearly abolished at the onset, not being regained in the other. In 3 no benefit resulted; in 1 the treatment was discontinued on account of pain, and in another on account of difficulty of application from the stoutness of the patient.[1]

See the accounts of M. Passavant's and Mr. Lansdown's cases given in the Appendix. The success of Mr. Lansdown's case raises the question whether ligature of the dilated ophthalmic vein might not be of service even in cases of undoubted arterio-venous aneurism behind the orbit.

[1] In Gioppi's case, digital compression was employed on the left side in four ways. 1. Strong pressure between the heads of the sterno-mastoid in a direct line from before backwards diminished the pulsation of the carotid, but prevented the return of blood by the anterior jugular vein, and occasioned some cyanosis. 2. By pushing the index, the middle, and the ring finger along the external and the thumb along the internal border of the sterno-mastoid, immediately below the crossing of the omohyoid, and squeezing it, after having pushed it posteriorly against the above-mentioned muscles, whilst the other hand placed upon the sinciput turned the head downwards and to the left to relax the tissues, the sheath common to the jugular, carotid, and vagus was encountered. The jugular vein and nerve could be made to glide posteriorly between the fingers, whilst the carotid artery was kept between them, and almost perfectly compressed. This method was very efficient. 3. By placing the index finger of one hand along the internal border of the sterno-mastoid in the superior cervical triangle, and pushing it backwards, the carotid was found, so that it was possible to compress it posteriorly upon the vertebral column. When the compression succeeded completely it was only momentarily, because the artery glided easily to the internal or external side, and then—

Of the 11 traumatic cases only 1 was cured, in 1 the pain and giddiness produced rendered it intolerable. In 1 the treatment was discontinued because it caused discomfort, in the remaining 8 no benefit resulted. Perhaps the case which was cured, M. Galezowski's, should be reckoned as idiopathic, as the injury had been sustained three years previously and had not been followed by any symptom, such as noise in the head, indicative of the commencement of the affection. If this be a correct view we should have 3 out of 6 idiopathic cases cured by digital compression and 7 traumatic cases deriving no benefit, a significant indication of a difference of pathological condition.

Digital compression may be either continuous or intermittent, and it is a circumstance specially worthy of attention that in the three successful cases it was only practised for a short period in the twenty-four hours, and not for many minutes at a time. Thus, in Gioppi's case as compression caused faintness it was only employed for a minute or two at a time by the convalescent patients in the ward, the patient herself and the pupils. Pulsation and noises ceased at the end of the fourth day.

In Scaramuzza's case digital compression employed very cautiously on account of aortic disease was practised for not more than five minutes at a time. It was maintained for eighteen days twenty or thirty minutes every day in five or six turns, the total period of compression being only seven hours twenty minutes. The eye had then entirely re-entered the orbit, and pulsation had ceased. In M. Galezowski's case compression was practised every two or three days for fifteen or twenty minutes, afterwards increased to forty-five or sixty minutes daily. Marked relief followed each sitting, and at the end of a month the movements of the globe had returned, the eyelid was raised, and the chemosis was gone. An interruption to the treatment for about a fortnight then took place. Then it was recommenced, and continued for more

4. A slight compression towards the larynx or first rings of the trachea could be tried. The patient practised both the third and fourth manœuvres. 'Ann. d'Oculistique,' 1858, t. xi, p. 731.

than two months every two or three days. A month later the protrusion had almost entirely disappeared, the patient heard no bruit, and none could be heard on auscultation over the cranium. At the time of the report the treatment was being continued, but there was every prospect of a complete cure.

Galvano-puncture has been tried twice each time unsuccessfully, and each time in a traumatic case. M. Petrequin's patient died, but whether in consequence of ligature of the carotid or subsequent galvano-puncture is not clear. Mr. Holmes attributes the fatal result to galvano-puncture ('Lancet,' 1873, vol. ii, p. 257).

Injection of ergotine subcutaneously was practised in the case of Schiess-Gemuseus, and Socin, and produced nausea, vomiting, œdema, and increase of the tumour.

Injection of a coagulating liquid into the pulsating tumour has been practised four times. In two traumatic cases (Bourguet's and Desormeaux's) a solution of the perchloride of iron was used and effected a cure, vision being restored. In my own case, as previously stated, the quantity injected proved to be insufficient to effect complete coagulation, and therefore no positive conclusion can be drawn. In Brainard's case the lactate of iron effected a cure after the failure of ligature, but the eye was lost. Brainard had previously tried the effects of the lactate of iron in solution on a dog without ill effect, and he had also used it on the human subject injecting it into the veins at the bend of the elbow. Each of the veins into which it was thrown was obliterated after a time, and converted into a fibrous cord. He considers that while the perchloride of iron is a foreign substance causing coagulation of the blood, and is apt to be followed by gangrene and suppuration, lactate of iron is composed of elements which are natural elements of the blood, and acts by gradually causing thickening of the coats of the veins, converting them into fibrous cords, and thus obstructing the circulation.[1]

[1] Brainard, in the 'Lancet,' 1853, vol. ii, p. 162. He refers to experiments by M. Pravaz, M. Giraldés, and M. Debout in 'Revue [illegible]. de

Ligature of the common carotid has been practised 46 times, the right carotid having been tied 20 times, and the left 26 times. Of the 44 cases, 18 were idiopathic and 26 traumatic. Of the 18 idiopathic cases in which ligature was practised, 12 were affected on the right side, and 6 on the left; 3 died, and 15 recovered. Two of the patients who died were females advanced in years, and both had atheromatous arteries. One aged 65 died on the 16th day from secondary hæmorrhage and cerebral disturbance; the other aged 63 died in 52 hours from cerebral causes only. The third patient (Mr. Critchett's) succumbed some months after the operation from repeated hæmorrhages from the orbit, the disease being, in all probability, malignant. Of the 15 patients who recovered, 12 were cured of the aneurismal affection, vision being restored in 8, lost or not regained in 3, and not mentioned in the others. In 3 partial benefit resulted, vision being lost in 1, impaired in 1, and not mentioned in the other. Of the 26 traumatic cases, 3 died and 23 recovered. The ages of the patients who died were respectively 17, 22, and 40. One of the three was subjected to galvano-puncture, subsequently to ligature, and the cause of death is not stated; in the two other cases the cause of death appears to have been pyæmia. Of the 23 patients who recovered after ligature of one carotid only, 14 were cured of the aneurismal affection in the orbit subsequently to injection, vision being lost or not regained, and the bruit being noted as persisting in 3. In 6 the cure was in all respects complete. In 1 both ophthalmic and external carotid arteries were tied. In 2 the operation was unsuccessful; 2 were subsequently operated on the opposite carotid, and 2 [illegible]. In 4 partial benefit resulted. [illegible] nished but not removed in 3, [illegible] only in the other. In Velpeau's [illegible] removed altogether on the left [illegible] right.

Paris,' May, 1853. On this subject see a Th[illegible] des Anevrysmes par le Perchlorure de Fer.'

Taking the two sets of cases together, we have a total of 44 cases. Out of this number there were 6 deaths (1 death having no connection with the operation in my judgment), 5 failures to cure the disease, 7 partial successes, and 26 cures. Of the 5 cases in which the operation failed, 2 were cured by ligature of the opposite carotid, and 1 by injection. Of the 26 cases cured, vision was restored in 17, lost or not regained in 7, whilst the bruit persisted certainly in 3, and probably in several other cases not examined with the stethoscope.

The 2 cases in which both carotids were tied occurred in America. In 1 case the interval between the operations on the two arteries was thirty days, in the other fourteen months. Vision appears to have improved in the former case, but it had been lost and was not regained in the latter.

One or two points of interest deserve a few words of comment.

1. In reference to the *return of the symptoms after ligature.* It is worth while to remark that it may take place in three different ways. In cases of arterio-venous communication the symptoms may return on the same side from want or deficiency of coagulation of the blood in the ophthalmic vein, carotid artery, and cavernous sinus. Possibly in some cases the aperture of communication becomes temporarily obstructed by clot which is afterwards re-absorbed; or, secondly, the symptoms may return on the opposite side. Obliteration of the enlarged ophthalmic vein may have taken place on the side originally affected, but the arterio-venous opening still remains patent, and the arterial blood is pumped through the circular sinus into the cavernous sinus and ophthalmic vein on the opposite side. Thirdly, in cases of true aneurism, the affection which seems to have returned on the opposite side may in reality be the formation of an aneurism of the same kind *de novo,* and not a real return of the disease. It is not quite clear whether the return of the symptoms on the opposite side in M. Herpin's case was due to the second or third condition just mentioned.

2. *The causes of loss of vision* in cases of intra-orbital aneurism, both before and after treatment, are worthy of attention. In some traumatic cases vision might be lost at once, and not regained on account of injury to the optic nerve, as probably happened in Scott's case. In some idiopathic cases it might be lost in consequence of the pressure of a circumscribed aneurism inflicting damage to its structure from which it could not recover, whilst in others dependent on ruptured aneurism in the cavernous sinus, the circulation may be so obstructed that ulceration and even sloughing may occur. Pressure on the ophthalmic nerve may cause ulceration of the cornea, either directly or indirectly. Vision has been lost in cases treated simply, treated by digital compression, treated by injection, and treated by ligature. Thus vision was lost in Mr. France's case about eight weeks after the commencement of the affection and was not regained when a spontaneous cure resulted. In M. Gendrin's case, although the disease spontaneously subsided, aided perhaps by the exhibition of digitalis, the eye suppurated and the cornea softened and ulcerated. In Scaramuzza's case cured by digital compression, sight was lost. In Dalrymple's case cured by ligature within a few months of the origin of the disease, vision which had been abolished at an early stage was not regained. In Jobert's case the cornea ulcerated after ligature, and when Velpeau saw the eye some time afterwards the globe was in a state of advanced atrophy. In one of Mr. Nunneley's idiopathic cases, sight, little impaired at first, was lost before ligature and not regained afterwards. In one of Mr. Nunneley's traumatic cases which was ultimately cured after ligature, sight was lost. In Mr. Laurence's case, in which the carotid was tied within a month of the accident, vision was abolished. In Mr. Bowman's second case sight was not much impaired before the operation, but it was lost afterwards. The cornea ulcerated in Brainard's case after injection, and in my own case after injection and ligature. On the other hand, healing of an ulcerated cornea took place before ligature in Busk's, and after ligature in Hippel's case, and vision, nearly abolished in several cases before treatment,

was restored afterwards. The contradictory results obtained may, I think, be explained in great measure by the varying amount of coagulation which takes place in the carotid and ophthalmic arteries, and in the cavernous sinus and ophthalmic vein. In arterio-venous communication between the carotid artery and the cavernous sinus, it is not improbable that the eye may in some cases be dependent for its nutrition, either entirely or partly on the blood brought by the ophthalmic vein, the current through the ophthalmic artery being diminished, and perhaps now and then altogether abolished. In such a case, when coagulation occurs in the carotid and the cavernous sinus, the blood supply may be so much and so suddenly curtailed as to be insufficient for the preservation of the transparant media, and before the anastomosing channels can become available, irreparable damage to these delicate structures may be wrought. In my own case I believe that the ulceration of the cornea was due to the closure both of the ophthalmic vein and ophthalmic artery, so completely was all trace of pulsation abolished in and around the eye and its appendages. In Mr. Curling's case the cornea grew hazy after ligature only, and sight might have been lost, but fortunately the collateral circulation was re-established in time for its preservation. These considerations lead me to think that sight rests in all cases of intra-orbital aneurism on a precarious foundation, and that it is impossible to predicate positively whether the treatment recommended will succeed in preserving or restoring it in any particular case.

3. Lastly it is desirable to endeavour to form an estimate of the comparative value of the different methods of treatment. My own conclusions may be briefly stated as follows:

(*a.*) The *exhibition of such remedies as belladonna, digitalis, and veratrum,* are worthy of trial before a recourse is had to the more active of the surgical means at our disposal, and may be used as adjuncts to digital compression, rest and regulation of diet, and the local application of ice.

(*b.*) *Digital compression* is most likely to succeed in idio-

pathic cases dependent on true aneurism of the carotid or ophthalmic artery, and very unlikely to succeed in cases of free arterio-venous communication. Experience has shown that the carotid artery is not an artery very readily compressed with effect, especially by medical students.[1] Nevertheless it is worth while to give it a fair trial for several reasons. It appears to be free from danger when applied with due precautions: it may be applied by an intelligent patient by himself, aided by nurses and students; it may effect a cure when only employed for a brief period at a time, and for a few hours spread over several days; even if it does not effect improvement, it is considered by observers (*e. g.*, Mr. Hart, M. Legouest, Mr. Z. Laurence, &c.) to be a most valuable preparation for ligature by establishing anastomosing channels and preventing any disturbance to the cerebral functions subsequently to ligature, and it affords useful information as to the probable results which would follow ligature.

(*c.*) *Instrumental compression* is more difficult to apply

[1] M. Legouest gives a graphic account of digital compression by medical students, to whom, however, the difficulties experienced in applying the method are by no means confined. He says, "After forty hours' duration had had no other results than to diminish during the period of its application alone the uncomfortable feelings which the patient experienced in the orbit, the tension and vascularity of the intra-orbital parts, and to determine sharp pains at the point compressed, and at the same time stiffness in the shoulder, the symptoms of aneurism remained the same. It is true that it was far from having been made with precision. I assured myself several times that the bruit de souffle continued during its application, and that blood passed sometimes abundantly into the carotid unskilfully seized, and escaping the fingers of my assistants. These assistants, being only relieved every half hour, became fatigued. Their fingers were benumbed, and became displaced by the movements of deglutition executed by the patient. He elevated his shoulder, inclined his head, contracted his sterno-mastoid and trapezius to escape the pain of compression which the assistants increased, but in vain, with the view of rendering it more efficacious. In fine, among the great number of assistants employed, several had more zeal and good-will than dexterity." Half an hour is much too long a time for any one to keep up digital compression. Ten minutes or, at most, a quarter of an hour without relief is the longest time which ought to be allotted.

than digital compression, and more likely to do harm to the important nerves in relation with the carotid.

(*d.*) *Galvano-puncture* does not appear to be well suited for application to a sac formed by a thin-walled vein, and would be worse than useless in cases caused by the presence of a circumscribed aneurism behind the orbit.

(*e.*) *Injection of coagulating fluids* is adapted only for cases of arterio-venous communication, and can be used most conveniently when the pulsating veins are prominent at and beyond the orbital margin. It is more painful than ligature, and probably involves more risk to vision, partly because it may cause inflammatory mischief, and partly because the coagulation effected by it may be so abundant as to interfere with the requisite supply of blood for the maintenance of the ocular tissues. The risk to life has not been determined.[1]

(*f.*) *Ligature of the common carotid* is, beyond question, the means most generally applicable to cases of intraorbital aneurism dependent on morbid states of the arteries. Judging from past experience, the risk to life in healthy subjects does not appear to be great. Ligature should not, I think, be practised in cases where there is heart-disease or evident atheromatous degeneration, or in old people. The two cases operated on over sixty years of age succumbed; and if we deduct these as well as Petrequin's case, in which galvano-puncture may have been the real cause of death, and Critchett's case, in which death had no relation to the operation as far as I can judge, only two deaths have taken place in forty cases, or forty-two instances of ligature of the carotid, just 5 per cent. At the same time it must be remembered that there is a considerable per-centage of cases in which the operation has been either partially successful or altogether unsuccessful in curing the disease, or in which an apparent cure has been succeeded by a return of the symptoms.

[1] Injection of coagulating fluids in these cases stands, at present at least, on a very different footing from injection of a nævus, and the fatal results of the procedure applied to nævi cannot be fairly used in condemnation of it in the treatment of aneurismal varix.

Nor is it altogether unlikely that in some of the cases reported as cured, and seen for the last time a few weeks after the operation, the affection may have returned when the patient was out of sight. Taking these points into consideration, and remembering such cases as those of Erichsen, Collard, Holmes, Freeman, and others, a hurried resort to ligature cannot be advised, whilst it should be borne in mind that the Hunterian method is not regarded as the best method for treating aneurismal varix in other situations. On the whole, however, I am strongly disposed to agree with Mr. Curling and Dr. Morton, that in traumatic cases and in suitable subjects ligature should be practised early when vision is threatened.[1] It is true that if the arterio-venous communication be free ligature may fail, but the balance of results is very much in favour of ligature; and should the operation fail to cure the disease, ligature of the opposite carotid, and injection of a coagulating liquid, are still in reserve. The mere persistence of a bruit during digital compression need not in any way affect the decision. Experience has now shown that the patient may be to all intents and purposes cured by ligature, and yet, owing to some changed condition of the blood-vessels, a bruit generally of an altered character and of diminished strength will remain for a variable time. If exophthalmos congestion, paralysis, and pulsation can be removed, the bruit is of no account. Lastly, in regard to ligature of the opposite carotid after ligature of one carotid has failed, we must be guided by the special cir-

[1] "When there is danger of the eye being compromised, or even without this, when the disease is increasing and compression has failed, ligation should be resorted to."—Morton, 'Amer. Journ. Med. Sci.,' July, 1860, p. 41. Mr. Curling would not wait for digital compression, and his opinion is confirmed by the negative results of digital compression in the *traumatic* cases. Compression, however, seems valuable as a preparation for ligature. Mr. Curling's case and Mr. Joseph Bell's case illustrate the opinions quoted. In both these cases the patient had lost the sight of one eye, and the affection threatened the other. Ligature was successful in preserving useful vision in both instances, the dilatation of the pupil in Mr. Curling's case being counteracted by the use of a card perforated with a small opening. In the idiopathic cases digital compression should be applied perseveringly.

cumstances of the case; but if we find the symptoms relieved by compressing the unoperated carotid, the favorable experience of our enterprising American brethren would afford us encouragement to expect success from the procedure. In conclusion let me say that I have offered the narration of my case to the Society, because I believe that the record of cases of intraorbital aneurism, whatever may be the issue of treatment, will materially advance the knowledge of a class of affections which has not yet been thoroughly elucidated, and I have accompanied the narration with as much information as I have been able to collect, in the hope that the labours of future observers and investigators may be lightened.

CHRONOLOGICAL RÉSUMÉ OF CASES OF INTRA-ORBITAL ANEURISM.

I. IDIOPATHIC CASES.

1. *Date, Surgeon, and References.*

1809. Travers, Benjamin. 'Med. Chir. Trans.,' 1809, vol. ii, p. 1. "A Case of Aneurism by Anastomosis in the Orbit, cured by the ligature of the Common Carotid Artery." Two portraits, one before and the other after cure, at the end of the volume.

Sex, Age, Side affected, Origin.

F. 34. Left orbit. A healthy woman, mother of five children, some months advanced in pregnancy, after suffering pain for some days felt a sudden snap on the left side of the head, attended with pain and followed by copious effusion of serum into eyelids.

Symptoms and Progress.

Increase of pain; inability to raise head from pillow; gradual protrusion of globe; appearance of two pulsating tumours on inner side; upper had vibrating thrill, lower pulsated with arteries; constant noise in head in recumbent posture; motions of eyeball impeded; sight impaired. More than four years later when seen by Mr. Travers, hollow of orbit lost. For description of tumours, see ante, p. 233.

Treatment and Result.

Œdematous swelling round orbit reduced by puncture. Issue in temples; leeches; cold washes; no improvement. Moderate compression borne only for a short time, owing to pain. Ligature of carotid in two places. Noise in head at once ceased, and pain was numbed. On twenty-ninth day patient went home and attended to her duties, the tumour being less, pulsation feebler, and pain removed. Five months after ligature a miscarriage with copious hæmorrhage. Next day pulsation in upper tumour ceased. Two months later hæmorrhage from bowels. A few months later cured. Years afterwards Mr. Hodgson saw the patient, and no one could have told she had had anything the matter with her eye.

2. *Date, Surgeon, and References.*

1812. Dalrymple, William. 'Med. Chir. Trans.,' vol. vi, p. 3. "A Case of Aneurism by Anastomosis in the Left Orbit, cured by tying the Common Trunk of the left Carotid Artery."

Sex, Age, Side affected, Origin.

F. 44. Left orbit. "Of a delicate and sickly habit of body," pregnant with her sixth child, seized in the middle of the night with intense pain in

her left eyeball. Attack sudden, instantaneous. Hearing a noise as of the cracking of a whip, and feeling at the same time an extraordinary pain in the globe, she woke in great alarm and leapt out of bed.

Symptoms and Progress.

Ten or twelve hours afterwards eye became inflamed and eyelids swelled; acute pain over whole of left side of head; anguish at bottom of orbit scarcely to be borne. Next night abatement of extreme pain, increase of swelling, feeling as if the eye were forcibly driven upwards. During labour, seven weeks afterwards, a bright red tumour projected between the eyelids. Ptosis came on early, and she became totally blind on the left side. Nine months after the origin of disease symptoms worse, pain constant, acute, chiefly referred to bottom of orbit; noise in head unceasing, like the rippling of water, absolutely insupportable with head lowered. Proptosis, ptosis. Paralysis of muscles of globe; red chemosed pad of conjunctiva lying over everted lower lid; cornea transparent; pupil dilated and fixed. For description of tumours, see ante, p. 233. Strong pressure on carotid lessened, but did not stop, pulsation in upper tumours.

Treatment and Result.

Ligature of left common carotid in two places, artery being divided between the ligatures. Pulsation ceased in tumours. Eight days after ligature no pulsation; eye in the orbit. Patient cured, but sight not regained.

3. *Date, Surgeon, and References.*

1823. Guthrie. 'Operative Surgery of the Eye,' 1823, p. 158.

Sex, Age, Side affected, Origin.

"I have seen one case of true aneurism of the orbit which terminated fatally. The symptoms were similar to those above mentioned (cases of Travers and Dalrymple), but no tumour could be perceived; the hissing noise in the head could be distinctly heard. On the death of the patient an aneurism of the ophthalmic artery was discovered on each side, about the size of a large nut. The vena ophthalmica cerebralis was greatly enlarged, and obstructed where it passes through the foramen lacerum in consequence of a great increase in size which the four recti muscles had attained, accompanied by an almost cartilaginous hardness, which had been as much concerned in the protrusion of the eye as the enlargement of the vessels."

Treatment and Result.

"The disease existing on both sides prevented an operation on the carotid being attempted, to which, indeed, the patient would not have submitted."

4. *Date, Surgeon, and References.*

1829. Roux. 'Journ. Hebdom.,' t. ii, 1831, p. 119. Demarquay. 'Traité des Tumeurs de l'Orbite,' 1830, p. 306. 'Gaz. Hebdom.,' 1836, p. 631.

Sex, Age, Side affected, Origin.

M. 26. Right orbit.

Symptoms and Progress.

A pulsatile tumour gradually developed itself at the angle of the right eyebrow. An oculist to whom he applied made with a trocar a puncture in the centre of the tumour. Very black blood escaped from the wound, and the ocular globe, which was only prominent, began to be deformed.

Treatment and Result.

Roux was obliged to ligature the carotid. The operation was followed by violent pains in the wound and in the tumour. During many days the patient was in a state of agitation bordering on delirium. In the end there still existed exorbitism and pain in the tumour.

5. *Date, Surgeon, and References.*

1829-30. Warren, Dr. J. C. 'Surg. Obs. on Tumours,' Boston, 1837, p. 400. "Aneurism by Anastomosis."

Sex, Age, Side affected, Origin.

F. 18. Right orbit. Experienced a sort of crowding feeling at the anastomosis of the angular ophthalmic and frontal arteries accompanied by a pain so severe as to make her give up her work as a servant.

Symptoms and Progress.

Having remained idle for some months she entered the hospital about a year after the origin of the disease. She was a fat comely girl. At the inner angle of eye, just above lachrymal sac, was a small tumour as large as a hazel-nut; it had an active pulsation extending to the "surrounding arteries." The pulsations of the facial artery were very strong, and by compressing it the vibrations of the tumour were much lessened; skin over tumour slightly reddened, with increase of heat. Pressure on carotid, which had an increased pulsation, arrested pulse of tumour. Over carotid and facial arteries the saw-mill sound was heard with the stethoscope. Pressure on temporal artery produced no change.

Treatment and Result.

A branch of ophthalmic was tied, the facial divided below, the tumour allowed to bleed to 18 oz. and then compressed; on division of facial pulsation ceased, and the patient was relieved from her bad feelings. On removing compression a slight pulsation was perceived. Pulsation did not wholly cease. She went out and returned in four months, the pulsations of the arteries going to the tumour and of the carotid being very strong. She felt sometimes as if her head was flying off. Face and forehead red and swollen. Patient kept low, bled and took digitalis without benefit. Temporal artery divided. Right carotid tied, and pulsations ceased at once; those on left side slowly subsided. At the end of two months discharged cured.

6. *Date, Surgeon, and References.*

1835. Baron. 'Bull. de la Soc. Anat.,' 1835, p. 178. "Delens," op. cit.

Symptoms and Progress.

Another specimen of aneurism has been presented by M. Baron. The

tumour was situated on the carotid as it passed through the cavernous sinus. It appears to have been ruptured at that part, and a decolorised coagulum the size of an almond occupied the cavernous sinus. Is it not to this cause we must ascribe the varicose condition of the orbital veins which had produced considerable exophthalmos? A further support to the opinion is found in the very intense bellows murmur which the stethoscope detected when applied over the projecting eyeball.

7. *Date, Surgeon, and Reference.*

1825. Gendrin. 'Leçons sur les Maladies du Cœur,' t. i, p. 240. Abridged in Demarquay, op. cit.

Sex, Age, Side affected, Origin.

F. 32. Left eye. Remarkably lean, with disease of heart and great vessels, was seized one evening with violent pains in the left eye. The day afterwards she had the eye pushed forwards as out of the orbit, and sight on that side was lost.

Symptoms and Progress.

A fortnight afterwards symptoms were:—Eyelids swollen, red, and raised as if by a foreign body; eyeballs pushed forward and immovable; cornea transparent; pupil immovable and largely dilated; conjunctiva feebly injected; sight lost; finger laid on eyeball with lids closed detected an elevation synchronous with the arterial diastole; continual groanings; rotatory movement of head; hand carried constantly to left parietal and temporal regions. Very pronounced friction-bruit over orbit and right eye not in temporal or malar regions. Case complicated with heart disease, aortic disease, and paralysis.

Treatment and Result.

Digitalis (5 centigrammes for a dose). For effect, see p. 244. *Post-mortem.*—Ophthalmic vein varicose and distended with coagulated blood. A second external orbit vein also gorged. Internal carotid and ophthalmic arteries surrounded by adherent clot continuous with clot infiltrated into arterial tunics. See pa. 214 and note to p. 221.

8. *Date, Surgeon, and References.*

1839. Jobert (de Lamballe). 'Mémoires de l'Academie de Médecine,' t. ix, p. 57. "Observation de Ligature de l'Artère Carotide primitive pour obtenir la Guérison d'une Tumeur erectile de l'Orbite," &c. "Carron du Villards sur l'Exophthalmie," 'Annales d'Oculistique,' 1858, p. 122; and Demarquay, op. cit.

Sex, Age, Side affected, Origin.

M. 50. Right eye. One morning after a fit of coughing perceived that the right eye was injected and projecting from the orbit. No precursory symptom.

Symptoms and Progress.

When seen by M. Jobert, eye driven from orbit; movements nearly abolished; eye red, injected, too sensitive to bear light; chemosis; constant lachrymation; ocular globe could not be covered by the lids. For

description of tumour, see p. 234. A very fine trocar plunged into it gave exit to *arterial* blood. M. Carron du Villards saw the patient before M. Jobert, told him the nature of the disease, and advised ligature. He was prevented by illness from operating.

Treatment and Result.

Bleedings, mercurial frictions, refrigerants, and astringents of no effect; acupuncture caused increase of swellings and violent inflammatory action. Vision was lost. Ligature of right common carotid; pulsation and pains ceased at once and movements returned. Sight not regained. Eye ulcerated and atrophied.

9. *Date, Surgeon, and References.*

1841. Dudley, Dr., of Lexington, 'Amer. Journ. of Med. Sci.,' Jan. 7th, 1843, p. 173, "Aneurism within the Cranium." Mackenzie, "Diseases of the Eye," and Demarquay, op. cit.

Sex, Age, Side affected, Origin.

M. Right eye. Experienced at irregular intervals attacks of pain above the right eye, augmenting in force and frequency.

Symptoms and Progress.

Before the end of the second year the eye protruded considerably from the orbit. In another year the right temple, as well as the right eye, was the seat of a morbid prominence, and the pain was so violent that it occasioned delirium, one attack lasting fifteen days. Sufferings of the patient were intense and incessant until an abundant spontaneous flow of a yellow fluid from the nose brought marked relief. Pains afterwards increased whenever the nasal flux ceased. Objects at a distance not distinguishable with right eye. Deafness in right ear. The eye was half an inch more prominent than the other. The inferior and external part of the frontal bone, including the orbital portion and the outer half of the orbital arch, were deeply involved in the malady, the corresponding portions of the parietal, temporal, and sphenoid separated from the body of the bones were comprised in an enlargement occupying the temple and the side of the head. The bones of the head and face were separated at the external angle of the eye sufficiently to admit the end of the little finger into the site of the transverse suture. The whole mass gave to the touch the characteristic thrill of aneurisms, whilst the eye seen from the side was agitated with an alternate movement of advance and recoil corresponding to the pulsation of the heart.

Treatment and Result.

Ligature of right common carotid. Pulsation at once stopped, tension and swelling diminished. Three weeks after ligature, eye almost restored to the orbit; patient could see distant objects. Quite well at the end of a month. Six months after operation the noise and movements felt by the patient in the head ceased, and the patient was following his occupation as a smith.

10. *Date, Surgeon, and References.*

1844. Herpin, M., of Tours. 'Gazette des Hôpitaux,' 1852, p. 550,

"Tumeur erectile de l'Orbite gauche. Ligature de la Carotide primitive gauche. Guérison. Menace de récidive du coté droit. Réfrigérants. Guérison definitive." Reported by Dr. G. Triquet.

Sex, Age, Side affected, Origin.

F. 59. Left eye. The affection began with pain in the left temple and left eye, at first intermittent and slight, but gradually becoming more and more severe with decreasing intervals between the attacks. Sight impaired, and tumultuous pulsations.

Symptoms and Progress.

Movements of eyeball impeded; lachrymation; chemosis; pains became exacerbated; sight lost after manifesiations of diplopia; proptosis; pulsations continuous and insupportable; vertigo. On admission patient seemed rather reduced by suffering. Exophthalmos about half of globe; sclerotic and cornea normal; œdematous chemosis; vision nearly abolished on left side; lids raised, distended, folds completely effaced, and lids only covering half of the ocular globe. Marked bruit de souffle over temporal region, synchronous with the arterial pulse, disappearing in great part on compression of carotid. "The case," says M. Triquet, "was evidently either simple or malignant tumour, or aneurism of the internal carotid, or perhaps of the ophthalmic artery."

Treatment and Result.

Ligature of left common carotid. Bruit almost ceased. In a week the eye had almost returned into the orbit and recovered its functions. Nine months afterwards commencing exophthalmos and bruit on right side, and compression on the right carotid did not stop the bruit and pulsation; ice was applied constantly for three months, with complete success. Seven years later the patient continued well.

11. *Date, Surgeon, and References.*

1851. Haynes Walton. 'A Treatise on Ophthalmic Surgery,' p. 258, *et seq.*, Lond., 1853, described as "Aneurism by Anastomosis," plate given at p. 259. 'Med. Times and Gazette,' 1854, vol. viii, p. 185.

Sex, Age, Side affected, Origin.

F. 4 months. Right eye. Slight prominence of eye one month after birth. Sister of patient had had a nævus of scalp.

Symptoms and Progress.

At four months eye prominent; lids swollen; cheeks puffy; conjunctiva thickly set with bright red vessels. Pressure on eyeball lessened the protrusion for a few seconds, whilst crying rendered the eye more vascular and caused great temporary protrusion. In a fortnight increase of all the symptoms; pulsation not distinctly felt, at least Mr. Walton could not satisfy himself of it; however, several persons declared that they felt it. Arterial souffle heard with stethoscope over the eye, not heard in the other orbit.

Treatment and Result.

Cold lotion applied for three weeks without result. Ligature of right common carotid when the patient was four months and three weeks old.

In a few days protrusion began to diminish. Pressure was then applied by means of an elastic bandage round the head; a year after operation the eye was nearly in its natural position, and all the movements were perfect.

12. *Date, Surgeon, and References.*

1851. Gervasi. 'Intorno alla Legat. della Carotide Primativa Ecc. Spezia,' 1851, p. 132. See 'Della Chirurgia in Italia, &c.,' di Alfonso Corradi, Bologna, 1871, p. 131. Described as "Aneurism by Anastomosis."

Sex, Age, Side affected, Origin.

M. 34. Right eye.

Treatment and Result.

Right common carotid tied. Cure.

13. *Date, Surgeon, and Reference.*

1853. Aubry, M., of Rennes. 'Gazette des Hôpitaux,' 1864, "Tumeur erectile de l'Orbite; pulsations; bruit de souffle; erreur de diagnostic; dilatation de la veine ophthalmique," by M. G. Triquet.

Sex, Age, Side affected, Origin.

F. 31. Right eye. Had had typhoid fever four years previously. The affection began subsequently. She was of limited intelligence, and was unable to give any clear account of her case, but she had no recollection of any injury to her head.

Symptoms and Progress.

On admission face congested; exophthalmos, but functions of eye preserved. Tumour the size of a nut on inner side of upper lid, subcutaneous and of the colour of the skin. Another tumour divided into two parts by the tendo-oculi existed below it. These tumours were soft, fluctuating, disappeared under pressure, and returned again. With tips of fingers pulsation readily detected. Over palpebral region the ear perceived a bruit de souffle very distinct, intermittent, and synchronous, with the arterial pulse and ventricular systole. It was soft, prolonged, and might be more correctly designated as continued with reinforcements at the moment of contraction of the heart. An aneurismal tumour of the ophthalmic artery was diagnosed. The patient suffered from attacks of vertigo. She died rather suddenly a week after admission.

Treatment and Result.

Post-mortem.—All organs healthy, but the brain under surface of middle lobe softened; walls of cranium very thick and vascular. For appearance of vessels, see ante, p. 188.

14. *Date, Surgeon, and Reference.*

1855. Hussey, E. L., Oxford. 'Oph. Hosp. Rep.,' 1859-60, vol. ii, p. 127, "Protrusion of the Globe, with some Symptoms of Aneurism."

Sex, Age, Side affected, Origin.

M. 42. Left eye. Disease commenced quite suddenly. Being very tired with driving a waggon for four days and nights, and being half asleep, he was roused by something amiss with his team; jumping up, he felt deep-seated pain in his left eye and singing in his ear. From that time the

pain and noise continued. He passed some bloody urine and spat some blood for three or four days.

Symptoms and Progress.

When seen two years after the commencement of the disease he had dimness of sight in left eye; throbbing and pain deeply seated at the back of the eye, much increased on stooping. Exophthalmos forwards and outwards; conjunctiva very vascular; no tumour appreciable by finger. Appearance of eyelids natural. On compressing carotid eye could be returned to its proper position. The man smiled and said that his sight was clear, and that all pain and throbbing had ceased. On relaxing pressure the protrusion and other symptoms returned. Six weeks after being seen by Mr. Hussey he lost all power over upper lid; pupil moderately dilated and motionless; sight gone; globe no longer returned to its place on pressure on carotid, either on right or left side. Subject to epileptic fits, and subsequently had frequent fits and became paralysed. Some months later he lost the sight of the eye, which became enlarged and hard, and emitted a serous discharge.

Treatment and Result.

He died fourteen months after coming under observation, or three years and two months after the commencement of the disease. Some time before his death the globe lost all its natural appearance, and protruded from between the lids a large, hard, raw substance. Skin of upper lid ulcerated superficially. A stream of blood flowed almost without intermission over the cheek from a depression in the middle of the globe. Free epistaxis. Post mortem forbidden.

15. *Date, Surgeon, and References.*

1856. Gioppi, Prof. (Padua). "Anévrysme de l'Artère Ophthalmique guéri par la Compression Digitale," 'Annales d'Oculistique,' 1858, t. xl, p. 215, taken from "Giornale d'Oftalmologia Italiano," short notice in 'Archives Générales de Médecine,' 1858, t. xi, p. 731. Abstract in Mr. Hart's paper, 'Lancet,' 15th March, 1862, with a woodcut of the patient.

Sex, Age, Side affected, Origin.

F. 42. Left eye. During an effort of childbirth she felt an unusual rattling in the left eye. She perceived that the eye was driven from the orbit and pushed the eyelids forwards. In childhood the patient had suffered from rachitis, and was the subject of rachitic curvatures of the spine and pelvis.

Symptoms and Progress.

Rapid formation of serous chemosis, deep pulsation and buzzing perceived by patient in the cranial cavity. After the labour proptosis increased, sight diminished, and was soon lost. On entering hospital twelve days later left eye was immovable and so prominent that the superior eyelid distended upon the tumour was rendered entirely immovable, and hardly covered the superior segment of the cornea. So tense was the eyelid that it could be raised only by the finger; it was swollen and of a livid red

colour, the tarsal cartilage more pronounced and the veins dilated; the volume formed in the eyelid projected in front of the orbital arch. Beneath the arch in the cavity of the orbit a softer tumour, tortuous, cylindrical, easily compressible, and when compressed offering a slight pulsation. It was towards the inner wall of the cavity, had a calibre of four or five millimetres, and did not pass the orbital border; on raising the eyelid the structure of the eye would have been scarcely recognisable but for the cornea and iris; cornea rather hazy; ocular conjunctiva strongly infected; pupil much dilated and fixed; no pain; sensibility impaired. Pulsation and thrill perceptible on any point of conjunctiva together with the souffle characteristic of aneurisms; constant buzzing and roaring in the left ear, depriving the patient of sleep. Compression of carotid instantly stopped pulsations, souffle, and buzzings, and permitted the globe of the eye to re-enter the orbit on pressure. By the ophthalmoscope, pulsation of the retinal veins was discovered; at the same time two arteries recognisable by their clearer colour could be seen to give pulsations synchronous with the arterial pulse; yellow spot undiscoverable.

Treatment and Result.

Some punctures of conjunctiva relieved the tension and swelling. As pressure on the carotid for more than a minute or two occasioned profound syncope, ligature was contra-indicated. Treatment of Valsalva and Albertini with digitaline and ergotine, &c., had no effect. The symptoms increased. Digital compression was undertaken preparatory to ligature (see p. 245). At the end of four days pulsation, bruit, and noises ceased. In three months the eye was in the orbit with perfect movements and nearly perfect vision, objects appeared diminished. Noise in the head had returned and a true aneurismal souffle could be heard over the left eye and over the head.

16. *Date, Surgeon, and Reference.*

1856. Nunneley. 'Med.-Chir. Trans.,' vol. xlii, p. 173. Case 2.

Sex, Age, Side affected, Origin.

M. 38 Left eye. A pale and feeble woolcomber noticed a small swollen spot in the lower lid of left eye, with difficulty of opening lids.

Symptoms and Progress.

The swelling increased steadily for thirty-four weeks, at the end of which he consulted Mr. Nunneley. Lids so much swollen that he could not open them; conjunctiva greatly congested; sight very dim; little or no pulsation; confusion and bewilderment in head; no noise; auscultation apparently not practised; some proptosis. By pressure it could be made to recede. Pressure on carotid diminished congestion of eyelids.

Treatment and Result.

Left common carotid tied. Swelling of lids and congestion at once lessened. In rather more than a month eye had receded; congestion and chemosis gone; lids less swollen. Patient resumed work; shortly afterwards, as he was wheeling a barrow up the hill, he felt his eye suddenly become worse, the lids more swollen, and sight impaired. By rest these

symptoms subsided. Twelve months after operation quite well with good vision.

17. *Date, Surgeon, and References.*

1858. Scaramuzza, Dr., of Verona. 'Thèse d'Henry,' Paris, 1856. "Considérations sur l'Anévrisme Artério-Veineux," applied for, but said to be out of print. 'Annali Universali,' 1856. Mr. Hart's paper, 'Lancet,' 15th March, 1862. 'Arch. Gén. de Méd.,' 1858, t. xii, p. 731.

Sex, Age, Side affected, Origin.

F. Left eye. In weak health, and subject to palpitation. A few days previously, after violent access of fever, she felt an acute pain in the left orbit—something seemed to give way in the orbit. The eye enlarged, and the patient could only distinguish light from darkness.

Symptoms and Progress.

On admission into the hospital the left eye projected entirely beyond the orbit; the lids did not cover the ball; the eye was injected and red; the cornea dull; light hardly discernible; pulsation and thrill over orbit and left temple; dilatation of the heart and of the arch of the aorta.

Treatment and Result.

Digital compression (see p. 246). Cure complete; vision lost.

18. *Date, Surgeon, and References.*

1858. Nunneley. 'Med.-Chir. Trans.,' vol. xlii, p. 175. Case 3.

Sex, Age, Side affected, Origin.

F. 65. Left eye. Mother of fifteen children. After returning from a walk was stooping down to take off her shoes when suddenly, as the crack of a gun, she felt something give way in the left eye. Instantly great pain and buzzing noise in the head; confusion and deafness on the left side. Eyeball protruded, was red and swollen, and felt as if it would burst. The lids became swollen and nearly closed.

Symptoms and Progress.

Six weeks later left eyelids quite closed, much distended, and greatly congested, with superficial veins. Eyeball considerably protruded; iris motionless; lens muddy; conjunctiva greatly chemosed and scarlet, with large convoluted veins; sight gone; decided pulsation synchronous with the pulse; distinct bruit; both bruit and pulsation most marked at the inner side of the orbit; a perpetual beating and buzzing noise in the head, causing confusion and bewilderment, much increased on lying down, when she felt as if the eyeball would burst; insomnia.

Treatment and Result.

Left common carotid tied. Pulsation, bruit, noise, distress, ceased at once. Death on sixteenth day. For post mortem see p. 214.

19. *Date, Surgeon, and References.*

1858. Carron du Villards. Annales d'Oculistique, 1858. Demarquay, op. cit., p. 293.

Sex, Age, Side affected, Origin.

F. 50. Right eye. Affection attributed to efforts made during her last confinement at the age of forty-five.

Symptoms and Progress.

Auscultation detected an intra-orbital pulsation synchronous with the movements of the heart. Compression of the carotid arrested all pulsation in the tumour, and lessened it. The pulse intermitted at every fourth beat. The patient had also aneurism of the right femoral artery.

Treatment and Result.

M. Carron du Villards abstained from expressing a decided opinion as to treatment. He lost sight of the patient, and heard some time afterwards that she had died suddenly.

20. *Date, Surgeon, and References.*

1859. Nunneley. 'Med.-Chir. Trans.,' vol. xlii, p. 187. Case 4. Also vol. xlviii, p. 28.

Sex, Age, Side affected, Origin.

F. 42. Right eye. Mother of six children. A week before confinement with seventh child got up as usual early in the morning, but owing to great and peculiar pain in the right side of the head was obliged to go to bed again. While lying there she suddenly—"sudden as a flash of light"—called out to her mother, who was standing by her bed, "The pain has gone into my right eye, it feels all on fire." The eyelids closed. Sight was not affected. Immediate sickness and faintness from pain, and confusion in the right side of the head and ear.

Symptoms and Progress.

A fortnight afterwards she was delivered of a healthy male child at full term. Increase of symptoms. About a fortnight later, when seen by Mr. Nunneley, the symptoms were almost complete proptosis of the right eye; sight quite lost; cornea hazy; iris immovable and moderately dilated; lids rather livid, and so greatly distended as to be unable to meet, the intervening space being filled with a thick, protruding, transverse fold of conjunctiva intensely congested and chemosed; great heat of the part, and a feeling as though the eyeball would burst;" over the right side of the head, and a constant noise in the ear just like, and as loud as, the steam hammer of a foundry." Bewilderment increased by exertion. Bruit heard, and pulsation felt, over the eye and also over the left temple, neither being so marked as in other cases. No fluctuation; tumour too solid either for fluctuation or fluid.

Treatment and Result.

Ligature of right common carotid. Instant cessation of noise in head and bruit; proptosis and congestion diminished. At the end of seven weeks could just discern light from darkness. Paralysis of muscles of orbit and iris continued, but eyeball receded and cornea became clear. Death about five years afterwards. For post-mortem appearances see p. 214.

21. *Date, Surgeon, and References.*

1860. Syme, James. 'Observations in Clinical Surgery,' p. 161. Reported also, with remarks, by Dr. Joseph Bell in 'Edin. Med. Journ.,' 1861, p. 1064. "Case of Pulsating Tumour in the Orbit cured by the Ligature of the Common Carotid Artery."

Sex, Age, Side affected, Origin.

F. 22. Right eye. Always enjoyed excellent health. In October, 1859, she noticed a slight tendency to protrusion of the eye. She married in December. Four or five weeks afterwards she suffered from urinary irritation, pain after and during micturition, and obstinate constipation and derangement of digestive system. She then suddenly felt a smart pain in her right temple, which lasted a couple of hours. She was ordered by her physician to rub the temple with a mixture of chloroform and laudanum, and during the rubbing felt a crack as if something had given way inside.

Symptoms and Progress.

A week after the first occurrence of morning pain double vision and proptosis. Poultice ordered under supposition of abscess. Two days later pain recurred with great intensity, followed by pain in the head, which continued throughout. Some weeks later conjunctiva of lower lid became everted and protruded, forming a prominent red mass below the eyeball. Several months afterwards when admitted into the Edinburgh Infirmary patient had an anxious, care-worn look; right eyeball was excessively prominent, as if it would burst from the confinement of the swollen lids; conjunctiva injected, tense, and œdematous; lids could hardly meet over eyeball; lower lid everted, with great vascular swelling of the conjunctiva. Between eyeball and margin of the orbit a soft pulsating tumour could be felt through the lid, and seemed to contain fluid. It was less like an aneurismal sac than a bundle of small vessels; it was compressible, and could be diminished in size, but not completely emptied. Pulsation accompanied by a sensible and audible thrill, very distinct at the margin of the orbit. On auscultating the head a most remarkable whizzing noise, audible and very annoying to the patient, audible to the bystanders at the distance of a yard. Restless night, the pulsation and whiz constantly waking her with a start. Pressure on carotid stopped pulsation, bruit, and noise, and the orbital tumour almost subsided.

Treatment and Result.

Scarification of conjunctiva and two salivations of no effect. Right common carotid tied with instant relief, diminution of tumour, and cessation of pulsation. A strip of conjunctiva removed with scissors at the operation, and the rest a few days afterwards. Eye in orbit in a week. Within a month patient discharged cured.

22. *Date, Surgeon, and References.*

1861. Bowman, Wm. 'Med. Times and Gazette,' August 4th, 1860, and July 27th, 1861. "Pulsating Tumour of the Orbit; Ligature of Common Carotid; Recovery." 'Royal Lond. Oph. Hosp. Rep.,' vol. iii, No. 2, New Series, p. 235. Reported by Mr. Francis Mason.

Sex, Age, Side affected, Origin.

F. 41. Right eye. Mother of six children. When six months pregnant was engaged in washing. Suddenly a pain came on with a blowing noise on the right side of the head, followed in a few hours by proptosis. She was confined three months afterwards.

Symptoms and Progress.

When seen a month later the symptoms were—proptosis about a quarter of an inch; hollow under orbital arch filled; pulsation easily felt with fingers; a decided sharp blowing bruit audible over whole of right side of head and face, and also on left side above the globe, loudest over left eye, fainter over the cheeks, and least of all over the forehead and temples, the sound being propagated most readily along the soft parts in the course of the vessels. Paralysis of external rectus only; convergent squint; diplopia; sight unimpaired; conjunctiva very vascular; fulness and engorgement of the upper lid; arrest of symptoms on compressing carotid. With ophthalmoscope choroid and retina were seen much congested. No headache or cerebral symptoms; no pain on applying pressure over the eyeball.

Treatment and Result.

Digital compression of carotid ordered by Mr. Hulke, ten to twenty minutes, three or four times a day for a fortnight. Symptoms increased. Ligature of carotid with relief to the symptoms. Paralysis of sixth nerve permanent. Eye still prominent. Forty-five days after operation hæmorrhage and collapse. Eye more prominent, and eyelids, &c., œdematous; appearance of pulsating swelling. Eyeball hard and prominent; much pain. Pulsation ceased on compressing left carotid; soft bruit heard over orbit. Ultimately able to follow her occupation, swelling subsiding, but vision lost.

23. *Date, Surgeon, and References.*

1861. Clarkson, Freeman, M.D., President of the Halton Medical Association, Milton, Canada, West. 'Amer. Journ. of Med. Sci.,' July, 1866, p. 277. "Intra-orbital Aneurism treated by Compression."

Sex, Age, Side affected, Origin.

M. 61. Left orbit. Pain in the eyeball. It subsided, and returned in a few months. Then both eyes became swollen, and the left projected. Double vision.

Symptoms and Progress.

Aneurismal tumour within and on nasal side of orbit, strongly pulsating, and with distinct whizzing bruit; tumour felt elastic and cyst like. He could see better without his glasses with his left eye than with them with his right. Slight stiffness and numbness of the left cheek; unable to breathe freely through his left nostril, and when lying on his right side both nostrils were closed. No pain, but stuffing, as if from cold; slept well.

Treatment and Result.

Application of cold and direct pressure against the swelling by a curved spring attached to an india-rubber band around the head. Digitalis in small doses. After a few weeks tumour became hard and dense, and ceased to pulsate; eye retired and vision improved. Five years later no reappearance of disease.

24. *Date, Surgeon, and Reference.*

1864. Nunneley. 'Med.-Chir. Trans.,' vol. xlviii, pp. 20 and 37. Case 2. "On Vascular Protrusion of the Eyeball."

Sex, Age, Side affected, Origin.

F. 47. A short stout woman, soon after getting out of bed one morning, was seized with a giddy fainting fit and something queer in the head. Immediately afterwards eye began to be affected, and gradually got worse.

Symptoms and Progress.

Three months later proptosis; lids red and tumid; congestion and chemosis of conjunctiva; sight dim; pupil dilated and sluggish; pulsating eyeball; noise in ear and giddiness in the head; throbbing; stretching pain in orbit. Symptoms relieved by pressure on carotid.

Treatment and Result.

Declined operation. A year afterwards unfit for work. Suffered much pain; frequently in bed. Less prominence and chemosis; pupil inactive; ptosis; useful vision lost; light painful. Eye kept constantly covered with a wet cloth.

25. *Date, Surgeon, and References.*

1865. Morton, Dr. T. G., Surgeon to the Pennsylvania Hospital. 'Amer. Journ. of Med. Sci.,' vol. xlix, April, 1865. "Aneurisms, with the History of a Case of Aneurism of the Ophthalmic Artery successfully treated by Ligation of the Common Carotid."

Sex, Age, Side affected, Origin.

F. 36. Right eye. Mother of four children. Two months pregnant. Was awakened from sleep by the report of a pistol overhead, but was persuaded that it was only a dream. A peculiar sensation in the head with a slight purring noise prevented her from sleeping.

Symptoms and Progress.

Dull headache and defective vision followed. Vision failed; eye became prominent; noise distressing; convergent strabismus. Seven weeks after confinement proptosis; congestion; vessels over side of nose tortuous and pulsating. Pulsation of eyeball seen and felt; aneurismal thrill; condition unendurable.

Treatment and Result.

Digital compression too painful. Ligature of right common carotid with immediate relief. In less than a month went home perfectly well. Vision excellent. Several years later continued well; no impairment of vision; had been confined without difficulty.

26. *Date, Surgeon, and Reference.*

1866. Œttingen, Dr., of Dorpat. "Zur Casuistik und Diagnostik der Orbitaltumoren." 'St. Petersburger Med. Zeitschrift,' Bd. xi, 1860. 'Klin. Monat. f. Augenheilkunde,' 1874.

Sex, Age, Side affected, Origin.

F. 64. Right orbit. Mother of five children. Three weeks previously

lesion excited by cold. Pains and prickings on right side of head. Proptosis and loss of sight. Thoracic and abdominal organs sound. No marked degeneration of arteries.

Symptoms and Progress.

Singing noises in head had disappeared; eyelids infiltrated; orbital hollow obliterated; lower lid everted; veins of eyelids not particularly distended; chemosis; anæsthesia of conjunctiva; pupil dilated and fixed; total loss of vision; exophthalmos; pulsation of eyeball; diastolic bruit; pulsation specially marked between globe and outer part of orbital floor, but felt all round the eyeball; bruit only audible over the eye. Pressure on right carotid stopped pulsation and bruit, but scarcely affected tension of parts. Ophthalmoscopic examination prevented by opacity of vitreous.

Treatment and Result.

Leeches, scarifications, compressions, with little effect. Pus in eyelids evacuated. Death two years afterwards; no arterial change. Partial obliteration of the orbital veins.

27. *Date, Surgeon, and References.*

1868. Wecker and Richet. 'Annales d'Oculistique,' 1869, t. 61, p. 186. "Sur les Tumeurs pulsatiles de l'Orbite." 'Heidelberg. Oph. Geseles.' 1868.

Sex, Age, Side affected, Origin.

F. 63. Left eye. On returning from a drive experienced a strong buzzing in the left ear. Was seized with shivering and violent headache, and was forced to lie down. The day after ptosis and absolute paralysis of motor oculi.

Symptoms and Progress.

A month later paralytic symptoms had diminished. One of the vessels of the orbit running along the forehead gave a perceptible thrill to the finger; exophthalmos; intense hissing bruit synchronous with pulse; noise in the head. During next two months proptosis and paralysis, and size of distended frontal vessel increased; slight pulsations were observed in the eye; retinal veins distended, and outlines of papilla effaced; a little later cerebral symptoms; sensation lost in right side of body; buzzing in the ear continual, depriving patient of sleep. She could only sleep in a voiture when the noise in the streets was stronger than that in her head. Dilated left ventricle.

Treatment and Result.

Digital compression arrested the pulsations, bruit, and buzzing for a time, but the excessive stoutness of the patient did not permit the constant application of Luer's compress. Ligature of left common carotid by Professor Richet. Noise in head ceased immediately, but a slight souffle could be heard on auscultation. Three hours after operation patient was struck with paralysis of right side, and fell into a state of stupor. Death in fifty-two hours. For result of post mortem see pp. 197, 215, and 223.

28. *Date, Surgeon, and Reference.*

1869. Morton, Dr. T. G. 'Amer. Journ. Med. Sci.,' July, 1870, p. 44.

"Aneurism by Anastomosis with Orbital Tumour. Involvement of the entire Side of the Head and Face by the Extension of the Disease."

Sex, Age, Side affected, Origin.

M. 25. Left eye. Soon after birth the left side of the head and face was observed to be rather more fully developed than the right. As far back as he could remember the left eye had been prominent. Many years ago he noticed a rushing sound in his head and eye, more intense at times, the protrusion and noise being increased by heavy work or the stooping posture.

Symptoms and Progress.

Excessive exophthalmos; vascular and chemosed conjunctiva; intense pulsation and marked thrill perceptible to the touch on the temple and eyeball; the noise could be heard at some distance from the head; a considerable tumour, irregular, flattened above, to be felt deeply in orbit and at the inner angle of the eye, the thrill being very marked and intense in the tumour itself; orbital portion of tumour globular and readily compressible, but with removal of pressure it filled instantly; movements of eyeball lost; fingers could be counted with great difficulty at one foot; marked continuous bruit in vessels of face; left side of tongue enormously hypertrophied; this varicose aneurismal condition invaded the entire side of the head.

Treatment and Result.

As pressure on the left carotid controlled all bruit and thrill, with immediate lessening of the orbital tumour and exophthalmos, ligation of the vessel was advised as the affection was increasing, and the risks of hæmorrhage were thought to be considerable.

29. *Date, Surgeon, and Reference.*

1869. Morton, Dr. T. G. Op. cit. "Aneurism by Anastomosis involving the Orbit at the Inner Angle."

Sex, Age, Side affected, Origin.

F. 12. Right orbit. Tumour growing gradually since infancy at the angle of the right orbit.

Symptoms and Progress.

Growth rather more than an inch in diameter; larger at times and more prominent under excitement; pulsation slight, and sometimes scarcely to be felt. The child said she felt or heard a sound in her head all the time. Tumour could be forced by pressure almost away, after which it gradually refilled; the exertion of going upstairs producing great prominence. The eye was not displaced, nor vision interfered with.

30. *Date, Surgeon, and Reference.*

1869. Galezowski. 'Traité des Maladies des Yeux,' 1872, p. 829.

Sex, Age, Side affected, Origin.

F. 60. Both eyes. Had existed five weeks in right eye, and three in left. No account of mode of origin.

Symptoms and Progress.

Eyes very prominent; eyelids fallen; serous chemosis. In right eye

pulsation of central artery of retina. On applying the ear to the two eyes a *bruit de souffle et de sifflement*. The patient herself heard the noise constantly in the ears, and compared it to that of a railway. There was probably a spontaneous fissure of one or both carotids.

31. *Date, Surgeon, and References.*

1871. Schmid, Dr., of Odessa. 'Klin. Monat.,' ix, 219, 1871. 'Annales d'Oculistique,' 1872, vol. lxviii, p. 245.

Sex, Age, Side affected, Origin.

M. 25. Right eye. Bleeding from the mouth one evening so abundantly that he lost consciousness.

Symptoms and Progress.

Six months later very pronounced exophthalmos; ptosis; pad of conjunctiva concealing lower lid; bulb injected; cornea a little troubled; iris hyperæmic; retinal vessel dilated and sinuous; visual field free; fingers could be counted at a distance. Pulsation and thrill to be felt in upper eyelid, weakened but not ceasing entirely on compressing the right carotid or even both carotids. Bruit de souffle at the superior border of the orbit, and over half the head. *Diagnosis.*—Aneurism of the ophthalmic artery at its origin from the internal carotid.

Treatment and Result.

Ligature of right common carotid. Pulsation and thrill ceased; bruit altered, but not abolished. Patient left hospital about five weeks after operation. He could count fingers at twelve feet; still slight exophthalmos and congestion; neither pulsation nor thrill, but the whistling or piping bruit could be heard occasionally.

32. *Date, Surgeon, and Reference.*

1872. Julliard, of Geneva. 'Note sur un Anevrisme Intra-orbitaire, &c.," Paris, 1873.

Sex, Age, Side affected, Origin.

F. 69. Left eye. Always had good health. Was sitting at work when she suddenly felt a violent pain in the left eye and temple, and lost consciousness for some moments. In the night the pains persisted, and the patient perceived that her eye enlarged rapidly, and that the sight was gone.

Symptoms and Progress.

The next day, 26th April, when seen at the hospital, symptoms were—exophthalmos, the eye forming a large tumour half the size of the first; superior eyelid deep red, strongly stretched, and œdematous; conjunctiva red, swollen, gorged with blood; cornea transparent; pupil easily distinguished, immovable, and dilated; the eye pushed outside the orbital cavity; passed by a centimetre at the least the superior eyelid; the cornea reposed upon the cheek a little below the cheek-bone. The tumour presented pulsations synchronous with those of the heart, easily perceived when the hand was applied upon it, and augmented when the head was lowered; no thrill and no bruit to he heard over the tumour with the stethoscope; much pain and very distinct beatings felt by the patient, as well as a constant buzzing in the ear. Atheromatous arteries and heart

disease; marked whispering bruit continuous with redoublements heard next day. Compression of carotid stopped the pulsations. High fever. Tumour formed by proptosed eye and swelling round it compared to the tumour in Freer's case.

Treatment and Result.

Ice and digitalis. Next day cornea tarnished, conjunctiva mortifying. April 28th.—The entire eye sphacelated; intense headache. May 3rd.—Eschar becoming detached; pulsations and bruit as before. 8th.—Pulsation and pain less; bruit not heard. 20th.—Tumour less; eye partly eliminated, now the size of a hen's egg, hard, and not painful; pulsations gone. June 2nd.—Pains and beatings in the right eye; left eye eliminated. 6th.—Ptosis on right side. 24th.—Patient left hospital quite well. Orbital tumour still the size of a hen's egg; eyelids œdematous. February 20th, 1873.—Exophthalmos gone; left eye not sunken; no pulsation in orbit, kern to conjunctivitis.

33. *Date, Surgeon, and Reference.*

1807. George Freer, Birmingham. 'Observations on Aneurism and some Diseases of the Arterial System,' p. 32. "Fungus Hæmatoides."

Sex, Age, Side affected, Origin.

M. 30. Left. Attacked immediately after an inflammatory fever with stiffness and throbbing in the orbit of the eye.

Symptoms and Progress.

Pain rapidly increased, and in a few days the eye was protruded and suppurated. Tumour daily increased, very minute vessels ramifying on its surface, and in two months was nine inches in circumference. Repeated hæmorrhages very profuse and restrained, with difficulty. Health gave way; dropsical symptoms and death.

Treatment and Result.

Large doses of opium alone gave relief. Astringents and escharotics applied, but too painful.

34. *Date, Surgeon, and Reference.*

1854. Critchett, George, Assistant Surgeon to the London Hospital. 'Med. Times and Gazette,' Dec. 23, 1854, and May 5th, 1855, "Aneurism by Anastomosis."

Sex, Age, Side affected, Cause.

M. 35. Right.

Symptoms and Progress.

Exophthalmos. Pulsation.

Treatment and Result.

In November, 1854, ligature of right common carotid. Patient went on quite well for several weeks; eye sloughed. Hæmorrhage from the orbit occurred at the end of a month; recurred several times, and the patient died in March; no cerebral symptoms; no examination of the body.

II. TRAUMATIC CASES.

1. *Date, Surgeon, and Reference.*

1830. Warren. 'Surgical Operations on Tumours,' Boston, 1837.

Sex, Age, Side affected, Cause.

F. Age not stated. Right eye. Fell down stairs and struck inner angle of right eye.

Symptoms and Progress.

Pulsating tumour at inner angle of right eye, extending into orbit and affecting the vision of the eye.

Treatment and Result.

Right common carotid artery tied. No effect on disease.

2. *Date, Surgeon, and Reference.*

1834. Scott, Surgeon to London Hospital. Case related by Busk, 'Med. Chir. Trans.,' vol. xxii, and Curling, op. cit., vol. xxxviii.

Sex, Age, Side affected, Cause.

M. Boy. Right eye. Fell into a ship's hold.

Symptoms and Progress.

Concussion; proptosis; fixed globe and pupil. Vision lost at the end of a month; pulsation of the globe, which could be both seen and felt. A week later profuse arterial hæmorrhage from the nose during a fit of coughing.

Treatment and Result.

Pressure made on the globe for two days; was too painful to be borne longer; ligature of the right common carotid. Cure; vision lost.

3. *Date, Surgeon, and Reference.*

1835. Busk. 'Med. Chir. Trans.,' vol. xxii, p. 124. "A Case of Aneurismal Tumour of the Orbit, cured by tying the Common Carotid Artery."

Sex, Age, Side affected, Cause.

M. 20. Left eye. Severe blow on the right side of the head from the gaff of a vessel.

Symptoms and Progress.

Concussion; hæmorrhage from the right ear and unmistakable symptoms of fractured base of skull. Early symptoms were—swelling of left eyelids from serous effusion; immovable globe; fixed and dilated pupil; impaired vision; "inflamed" conjunctiva; onyx and ulceration of cornea, which subsequently healed. Six months and a half after accident, when on the point of being discharged, pulsation of the globe and a pulsating tumour detected; the latter was at the upper and inner corner of the orbit, immediately within the supra-ciliary ridge. Distinct thrill; loud, whizzing bruit; loud noises in head, on right side like church-bells, on left like breaking of waves on sea-shore. Pressure on carotid stopped bruit and pulsation.

Treatment and Result.

Having bled the patient the day before to 20 oz., Mr. Busk tied the left

common carotid; pulsation, bruit, and noise in head ceased. Four hours later feeble pulsation and whiz as loud as ever. Next day bled to 16 oz. with benefit, and the following day tumour, pulsation, and bruit all gone, and eyeball receding. Discharged in seven weeks and five days after operation. Cure; good vision with upper half of cornea.

4. *Date, Surgeon, and References.*

1839. Velpeau. 'Bulletin de Thérapeutique,' t. xvii, p. 128, 1839; 'Leçons Orales,' t. iii, p. 437; Art. "Orbite," Dict. en 30 vols., t. xxii, p. 323. Reported by Velpeau as "Erectile Tumour of the Orbit," the lesion being seated on the branches of the ophthalmic artery.

Sex, Age, Side affected, Cause.

M. 30. Both eyes, right more severely. Blow on nape from fist of a very muscular man.

Symptoms and Progress.

The day after accident right eye more prominent, and strong beatings in the head. Left eye became prominent after a time. Two months later dilatation of veins of eyelids, some of which became prominent and resembled cysts. Four months and a half after the accident there were pulsatile tumours and bruit on both sides; the tumours were situated beneath the orbital arch under the skin of the upper eyelid, and the pulsations were evident to sight and touch; bruit over orbits rasping, and louder on right side. See ante, page 186.

Treatment and Result.

Ligature of right common carotid; noise in head, pulsations, and bruit stopped at once and continued absent for some time, but at the end of three months they had all returned on the right side, and the tumour was reproduced. The patient declined further operation. Partial success.

5. *Date, Surgeon, and Reference.*

1845. Petréquin, Chirurgien-en-Chef de l'Hôtel-Dieu de Lyons. 'Comptes Rendus Hebdomadaires des Séances [illegible] Sciences,' 1845, t. xxi, p. 994; 'Gazette Médicale,' 1846. [illegible] Aneurism of the Ophthalmic Artery and of the Origin of its [illegible]

Sex, Age, Side affected, Cause.

M. 22. Left orbit. In October, 1841, fell [illegible] aches were the only primary symptom.

Symptoms and Progress.

Three months after the injury the patient [illegible] orbit, which presented the characters of an aneurism [illegible] with the arterial pulse; bruit de soufflé; [illegible] marked; sight weakened, but preserved. When [illegible] tumour was small, [illegible] pulsation and diminished [illegible]

Treatment and Result.

On the 5th of June, 1846, [illegible]

the symptoms. At the end of the second week bruit and pulsation returned. About a month later galvano-puncture was applied, in the hope, M. Petréquin observes, of saving the life of the patient. It was the first of M. Petréquin's cases treated in this way, and the mode of application left, he says, much to desire. The patient died in about a fortnight; a post-mortem was not obtained.

6. *Date, Surgeon, and References.*

1851. Brainard, Dr. Daniel, Professor of Surgery, Rush Medical College, Chicago. "Case of Erectile Tumour of the Orbit, cured by Infiltration with the Lactate of Iron after the Ligature of the Carotid Artery had failed, with Observations on the Effect of that Solution in operating on the Blood-vessels." 'Lancet,' 1853, vol. ii, p. 162.

Sex, Age, Side affected, Cause.

M. 34. Left orbit. Severe kick from a horse on the left side of the lower jaw, fracturing it on the right side and severely injuring him.

Symptoms and Progress.

On recovering from the shock he heard a blowing sound in his head; the eye became prominent, and pulsated with a thrill when touched; a loud blowing sound audible over the orbit and more or less over the head; arteries of head and neck pulsated with unnatural force; veins of face prominent; head hot; severe pain in head, with nausea and vomiting; pressure on left carotid instantly stopped pulsation and sound. At the end of four months much worse; more protrusion; conjunctiva ulcerated; lids incapable of closure; thrill heard over whole head; bilious attacks and vomitings had increased the size of the tumour; great heat of head and insomnia; only able to sleep by keeping cloths dipped in cold water applied constantly to it; anxious for operation.

Treatment and Result.

On account of great tenderness compression could not be borne. Ligature of carotid four months after accident; pulsation and bruit ceased; bags of pounded ice and evaporating lotions applied. On third day a slight pulsation and sound were detected; symptoms returned. A year after operation exophthalmos; eversion of lower lid, which was covered with a fungous projection; elastic and pulsating swelling at upper and inner part of orbit, near root of nose; small vessels of forehead and side of nose dilated and pulsated strongly with peculiar thrill. Puncture with hot knitting needles on three occasions. A drachm of solution of lactate of iron (8 grs. to ʒj of distilled water) injected into the tumour; intense pain in left temple, nausea, and vomiting resulted. For twenty-four hours all liquids were rejected, and the vomiting lasted six days; bladders filled with ice and salt gave relief to pain; neither thrill nor sound could be perceived after the injection; veins were much diminished and arteries reduced to normal state; ulceration of cornea, severe inflammation of the eye, and loss of the humours succeeded, and the eye collapsed. The patient resumed his ordinary occupation three months after the injection. Cure, with loss of eye.

7. *Date, Surgeon, and Reference.*

1852. Nunneley. 'Med. Chir. Trans.,' vol. xlii, p. 168, Case 1.

Sex, Age, Side affected, Cause.

M. Left eye. Blow on left eye whilst fighting.

Symptoms and Progress.

Beating noise in head and ear came on immediately and continued throughout. A few weeks after blow eyes bloodshot, sight dim, and aching pain; globe prominent; conjunctiva congested; iris sluggish; movements of globe slightly impeded; sight not materially affected. Two months later symptoms aggravated, and pulsation became perceptible, with a decided thrill. Pressure on carotid arrested it, and relieved the other symptoms.

Treatment and Result.

Puncture of tumour beneath upper lid caused increased swelling. Ligature of left carotid about four months after accident; pulsation and noise ceased and proptosis diminished. In three weeks the eye was nearly natural. The symptoms returned, then abated, and then returned again. Ligature of the right carotid was thought of, but after two or three venesections the patient improved. Ultimately he was cured, with loss of sight from a cataractous condition of the eye. In 1859 he remained well. Cure; vision lost.

8. *Date, Surgeon, and Reference.*

1853. France, Ophthalmic Surgeon to Guy's Hospital. 'Guy's Hospital Reports,' ser. iii, vol. i, 1853, p. 58. "Case of Pulsating Swelling in the Orbit."

Sex, Age, Side affected, Cause.

F. 38. Left orbit. Thrust in the left orbit with an umbrella from a drunken soldier.

Symptoms and Progress.

Ecchymosis. Eyeball protruded in a fortnight. Symptoms subsided, but a month later recurred. Inflammatory œdema of upper lid; lower lid everted and covered with projecting conjunctiva; injection of conjunctival vessels; veins of orbit distended; face swollen; vision lost entirely in eight weeks; pain so great as to preclude sleep. Pulsation on nasal and temporal sides of the eye, and as protrusion diminished a definite rounded tumour with pulsation appeared at the inner canthus.

Treatment and Result.

Mixture of acetate of ammonia, antimonial wine, and sulphate of magnesia; scarification of conjunctiva; chemosed pad incised; symptoms abated. As the swelling subsided the lower lid was brought into position with plaster. At the end of eight months the tumour and pulsation had disappeared, and the movements of the eyeball were regained, but vision was lost. Eighteen months later she continued well.

9. *Date, Surgeon, and Reference.*

1854. Curling, T. B., Surgeon to the London Hospital. 'Med.-Chir. Trans.,' vol. xxxvii, p. 226. "Case of Traumatic Aneurism of the Ophthalmic Artery consequent on injury to the head, cured by Ligature of the Common Carotid Artery."

Sex, Age, Side affected, Cause.

M. 49. Right orbit. Fell from the top of a stack of wood seven feet high on to his right shoulder and right side of his head.

Symptoms and Progress.

Evident symptoms of fractured base of skull. In five or six weeks congestion and chemosis of conjunctiva; eyeball became prominent; great pain on right side of head; pulsation detected on placing the finger on the upper lid and pressing gently on the globe; proptosis increased, and a very distinct bruit was heard over the right temple. Vision not impaired, but in a few days it begnn to become so, and other symptoms were aggravated: motions impaired.

Treatment and Result.

Having lost the other eye from cataract, it was of great consequence to the patient to preserve the right eye, so that the right common carotid was tied ten weeks after the accident; the pulsation and beating in the head was at once arrested. Next day vision was lost, and pupil dilated and fixed: chemosis. Congestion and protrusion subsided, but cornea became dull and hazy. It cleared, however, in a few days, and patient regained useful vision, and with the aid of a card perforated with a small hole was able to read with a little difficulty; power of moving eye was recovered.

10. *Date, Surgeon, and Reference.*

1854. Van Buren, Dr. W. H., Professor of Anatomy in Univ. Med. Coll., and Surgeon to the New York Hospital. 'New York Journal of Medicine,' July, 1857. In Dr. Wood's article on "The Early History of Ligature of the Common Carotid."

Sex, Age, Side affected, Cause.

M. 21. Left orbit. Fall of the walls of the house on which he was labouring.

Symptoms and Progress.

No external injury, but profuse bleeding from left ear, paralysis of left facial nerve, and concussion. Four weeks later protrusion of eyeball, tensive pain, and injection of conjunctival vessels; feeling of throbbing; aneurismal thrill discovered with stethoscope; pulsation and thrill arrested by pressure on left carotid.

Treatment and Result.

Ligature of left common carotid; pulsation, throbbing, and tensive pain arrested; exophthalmos subsided, but never entirely disappeared. Slight aneurismal thrill returned three weeks later, but it subsided. Patient was well when seen a year and a half afterwards. Cure; good vision.

11. *Date, Surgeon, and References.*

1855. Nélaton. Thèse d'Henry, 1856. 'Considérations sur l'Anévrysme Artério-Veineux' (out of print). Dr. Delens, op. cit. Holmes, "Lectures on the Surgical Treatment of Aneurism," 'Lancet,' 1873. Originally related, but briefly and with inaccuracies, in 'Bulletins de la Société Anatomique,' t. xxx, 1855. Short account by Dr. E. L. Holmes, of Chicago,

who witnessed it, in 'Amer. Jour. Med. Sci.,' July, 1864, new series, vol. xlviii. Gougenheim 'Thèse.'

Sex, Age, Side affected, Cause.

M. 21. Right eye. Thrust with the ferrule of an umbrella in the left lower eyelid.

Symptoms and Progress.

Abundant bleeding from nose and ptosis of right upper eyelid. Wound of left eyelid healed rapidly, but at the end of some days the right eye became more prominent, with diplopia and mydriasis. Two months later left eye healthy; right eye prominent; ptosis; dilated veins on surface of upper lid; pupil largely dilated; external strabismus; absence of movement of the eyeball; diplopia and presbyopia of right eye, which had been myopic before; patient blew blood habitually from the right nostril; paralysis of right third pair; pulsation of eyeball; bruit de souffle continuous, with reinforcements during arterial pulse; absence of thrill. A little later "bruit de piaulement" audible also to the patient. The patient continued to blow blood from the right nostril, and at times had epistaxis. See anté. page 189, for M. Nélaton's diagnosis.

Treatment and Result.

Compression of the right common carotid was adopted as the method of treatment, at first with M. Charrière's apparatus, which did not fulfil all the necessary conditions, and then with one devised by M. Henry. Compression did not occasion discomfort, but was ineffectual. The patient had already had rather abundant epistaxis on several occasions, and though he entered the hospital in order that the apparatus might be applied with greater accuracy, he had two returns of abundant epistaxis followed by syncope, and he died suddenly the day after admission. See ante, page 189, for result of post-mortem.

2. *Date, Surgeon, and Reference.*

1854. Bourguet, Chirurgien-en-Chef de l'Hôpital d'Aix. "Note sur un Cas d'Anévrisme d'Artère Ophthalmique et de ses principales branches gueri au moyen des Injections de Perchlorure de Fer" (lue à l'Académie des Sciences dans la Séance du 19 Novembre, 1855). 'Gazette Médicale de Paris,' 1855, p. 772.

Sex, Age, Side affected, Cause.

F. 12½. Right eye. Fell, three years previously to coming under treatment, from the second story of a house, and sustained two contused wound on the right side of the forehead, which suppurated for a month and a half or two months.

Symptoms and Progress.

Six months after the fall the parents noticed that the right eye became more prominent than the opposite eye, and that there existed a small pulsatile tumour at the internal part of the orbit, opposite the lachrymal sac. The tumour by degrees increased, and when M. Bourguet saw the patient there were a series of soft, indolent, elastic, pulsatile tumours at the base of the forehead, the inner part of the orbit, and in the upper lid; the frontal

tumour, as large as a large almond, reached to the middle of the forehead; the internal tumour was the size of a pigeon's egg, and the tumour of the eyelid was formed of a single trunk, the size of the little finger, and of numerous flexuosities and circumvolutions anastomosing together; all the tumours had a vibratory thrill; bruit de souffle continuous with redoublement, extremely distinct and audible even to the naked ear; skin over tumours thin and bluish; eye in great part driven from orbit, and the seat of pulsations synchronous with pulse; vision almost lost in right eye. Pressure on carotid abolished pulsation of tumours and eyeball and bruit.

Treatment and Result.

Electro-puncture (Bunsen's pile with six cells); four needles successively introduced into various parts of the tumour; great pain, but no clot formed. Three other sittings without effect. Injection of six or seven drops of solution of perchloride of iron to 28° into tumour; pulsations at once ceased in the part. Compression of both carotids maintained before and for twenty to twenty-five minutes after the injection, and cold applications were made to the forehead. The next day a second injection of seventeen to eighteen drops; some nausea and vomiting followed during the rest of the day. Next day tumours on forehead hard; eyeballs more prominent; eyelids infiltrated, the pulsation and bruit persisting in the upper eyelid and bottom of the orbit; then the prominence and pulsations gradually lessened. The patient left the hospital, and ten days later the tumours were all devoid of pulsation, and with a yellow tint like that of ecchymosis. At the end of ten months the eye completely retired into the orbit; the tumours had disappeared, vision was perfect, and the cure left nothing to desire.

13. *Date, Surgeon, and Reference.*

1857 to 1859. Gurdon Buck. Abstract given by Dr. Noyes. 'New York Medical Journal,' 1869, p. 664. Compare Poland, 'Royal Lond. Oph. Hosp. Reports,' 1859-60, vol. ii, p. 219.

Sex, Age, Side affected, Cause.

M. 22. Right eye. Fell from aloft on to the deck, and struck on his feet, ten weeks before admission into hospital in December, 1857.

Symptoms and Progress.

Insensible till following day, then found sight gone. Four weeks after pain commenced at inner angle of right eye, with throbbing and whizzing in the ear. On admission.—Marked exophthalmos, eye being pushed downwards and outwards; veins of upper lid enlarged and tortuous; conjunctival and scleral vessels dilated; pupil widely dilated and immovable; pulsation distinct and arrested by pressure on the carotid.

Treatment and Result.

Right common carotid artery tied shortly after admission; tumour did not subside, but pulsation was less marked; exophthalmos began to decrease, but all the symptoms returned, and on June 11th, 1858, the patient was discharged. He went to sea, and made a visit to London, returning in February, 1859; exophthalmos greater; eye scarcely any perception of light. Left carotid tied on February 23rd, 1859; a few minutes after

still a thrill in tumour, but less distinct. June 15th, protrusion nearly gone; perception of light only; a bruit audible at times in the tumour. Nov., 1859, bruit and tumour gone; vision nil; pupil enlarged.

14. *Date, Surgeon, and Reference.*

1857 and 1858. Halstead, Dr., New York. Case given by Dr. Noyes. 'New York Medical Journal,' 1869, p. 665.

Sex, Age, Side affected, Cause.

M. 37. Both eyes. Fell through a hatchway, Dec. 10, 1857, and was admitted into New York Hospital.

Symptoms and Progress.

After eight days complained of noise in left ear; left pupil sluggish; more contracted than the right. On 10th day diplopia; injection of both eyeballs. About two months after injury ptosis of right upper lid. A week later left eye began to protrude; chemosis in both eyes; distinct bruit heard on left temple and all over head, most decided over left frontal sinus; pulsation detected by pressure on eyeball.

Treatment and Result.

Feb. 14th, 1858, left carotid tied; immediately pulsation and bruit ceased and tumour diminished. Next day ptosis diminishing, noise in ear gone. On 20th noise in ear and bruit returned. April 3rd, discharged cured; no bruit; sight occasionally dimmed.

15. *Date, Surgeon, and Reference.*

1858. Bowman. 'Oph. Hosp. Rep.,' April, 1859, p. 6. "All the capital signs of orbital aneurism present in a marked degree, but independently of aneurism or any erectile tumour." By Mr. Hulke.

Sex, Age, Side affected, Origin.

F. 40. Left orbit. Blow from a fist on left side of head and temple, which knocked her down.

Symptoms and Progress.

Next day severe pain in left temple, disturbing sleep. At the end of a fortnight it was replaced by a rushing noise like the beating of a steam-engine; noise constant and audible to her husband; sight "bothered." At the end of four months and a half left eye became red and projected. On admission, on February 19th, into King's College Hospital, at the end of five months.—General fulness of orbital region; congestion; proptosis. Pupil dilated, but active; distant objects seen perfectly; inability to read. Loud sibilant bruit audible over left side of head; pulsation of eyeball; no head symptoms; depression at lower border of orbit between malar and upper maxillary bones.

Treatment and Result.

Left common carotid artery tied February 27th; pulsation and bruit at once ceased. Next day eye less prominent; sight better. March 7th.—Bruit heard over eye and a little above ear, a continuous musical note swelling out at each pulsation. 21st.—Ligature came away. Next day secondary hæmorrhage; eyeball again prominent; pupil dilated and fixed;

eye everted; ptosis. 15th.—More bleeding. 17th.—Death. For result of post-mortem see ante, p. 191. The malar was detached from the maxillary bone.

16. *Date, Surgeon, and References.*

1858. Hirschfeld, L. 'Comptes Rendus de la Société de Biologie,' 1859, t. v., 2 série, p. 138. "Épanchement de Sang dans le Sinus Caverneux du Côté Gauche, diagnostiqué pendant la vie." Dr. Delens, op. cit., p. 75. Holmes, "Lectures," 'Lancet,' 1873. 'Gazette des Hôpitaux,' 1859, p. 51.

Sex, Age, Side affected, Origin.

F. 78. Left. Fall from a carriage on to the pavement.

Symptoms and Progress.

A large wound at the root of the nose; very free bleeding. The wound healed a month later without any precursory symptom. She lost the power of raising the left upper eyelid and moving the eyeball. Some exophthalmos; complete anæsthesia of the eyelid, ala of the nose, and forehead; vision normal. Diagnosis on admission in this state into hospital two months after accident.—"Effusion of blood compressing the motor nerves before their entry into the orbit."

Treatment and Result.

9th January (second day after admission).—Small blister to left temple. 10th.—Erysipelas of face followed by intense fever; coma and death on the seventeenth day. *Necropsy.*—A blood-clot enveloping the third and compressing the ophthalmic nerve found in the cavernous sinus, and this clot covered a small circular hole in the carotid, which looked as if it had been punched out and was occupied by a string of decolorised clot about two inches long, passing into the mass of coagulum; bones sound.

17. *Date, Surgeon, and Reference.*

1859. Corner, F. M., Surgeon to the Poplar Hospital. 'Transactions of Hunterian Society,' 1874.

Sex, Age, Side affected, Origin.

M. 33. Right eye. Fall from aloft on to the deck of a ship, striking the left side of the head against the combings of the hatchway, in March, 1859.

Symptoms and Progress.

Insensibility, bleeding from ear, and afterwards deafness of left ear, due, it was thought, to fracture of the base of the skull. In May, when about to discharge him, Mr. Corner noticed a fulness of the right eyeball and pulsation at each beat of the heart; loud whizzing bruit audible over the head; bruit *double* and audible to patient. Pressure on carotid stopped bruit and pulsation.

Treatment and Result.

May 18th.—Ligature of right common carotid; symptoms at once arrested. Before the end of the day the bruit returned, but not the pulsation. Twelve

years afterwards he noticed that the bruit stopped for a minute or two and returned, then stopped for a longer time, and finally ceased entirely. The patient was shown to the members of the Hunterian Society. The cure was complete.

18. *Date, Surgeon, and References.*

1860. Passavant, de Frankfort sur Mein; Wecker, 'Maladies des Yeux,' t. i, pp. 803, 804.

Sex, Age, Side affected, Origin.

F. 9. Side not stated. Some knitting was thrown at her in a rage by her sister; one of the needles penetrated the external side between the eyelids, but was withdrawn.

Symptoms and Progress.

A short time afterwards an exophthalmos supervened with all the symptoms of an aneurismal tumour. As the eye had lost all mobility outwards, and was strongly turned inwards, it was thought that the affection was an aneurism of the lachrymal artery.

Treatment and Result.

Part of external wall of orbit resected, the soft parts having been dissected off the bones sufficiently to permit the finger to penetrate behind the globe. At the apex of the orbit, very deeply, and on the inner side of the optic nerve, the finger encountered the pulsations of an aneurismal pouch, but all attempts to tie it were in vain. Swelling of the lids and cheek supervened, but the wound healed, and the child ultimately remained in the same state as before.

19. *Date, Surgeon, and Reference.*

1861. Ernest Hart, Surgeon to West London Hospital. 'Lancet,' March 15th, 1862. "On a Case of Intra-Orbitar Aneurism cured by Ligature after Failure of Digital Pressure." Plate given.

Sex, Age, Side affected, Origin.

M. 11. Left orbit. Blow from the forked end of an iron rib of a parasol at the inner angle of the left upper eyelid.

Symptoms and Progress.

Free bleeding and rapid swelling of lid; the latter subsided, and eye continued a little bloodshot; headache and singing noises in head. Nearly four years after accident the mother noticed a pulsating tumour at site of wound in lid; this increased; lids became protuberant; conjunctiva congested; eyeball prominent. The tumour had strong pulsation and perceptible thrill; loud whizzing bruit audible all over left side of head and temple, continuous through systole and diastole; whizzing noise in head like a steam-engine. A large tortuous vessel could be felt between the eye and the roof of the orbit; this was thought to be the frontal branch of the ophthalmic artery. Pulsation arrested by pressure on the carotid.

Treatment and Result.

Intermittent compression by a staff of three persons of left carotid for

several hours daily, fifteen minutes at a time, during three weeks; pressure borne well; beneficial, but not curative. March, 1861.—Ligature of left common carotid; immediate cessation of pulsation and flattening of tumour. In 1862 no trace of tumour or pulsation; no noises in head; whizzing bruit still audible over the head. In all other respects quite well.

20. *Date, Surgeon, and Reference.*

1862. Greig, David, M.D., Surgeon to the Royal Infirmary, Dundee. 'Ed. Med. Journal,' Nov., 1862, p. 446. "Case of Intra-Orbital Aneurism cured by Ligature of the Common Carotid Artery."

Sex, Age, Side affected, Origin.

F. 47. Left orbit. Fell down a stair and struck the left side of her head against the framework of a loom.

Symptoms and Progress.

Stunned, confused and sick. The next day at work, but feeling "stounding" pains in her head and singing noise in left ear. Ten days later obliged to go to bed on account of severity of frontal headache, and she began to complain of her eye as well as of the singing noise in her left ear. Fourteen days after the accident, when admitted into the infirmary, she exhibited proptosis of left eyeball, which was nearly covered by the swollen, œdematous, livid, red, and protruded lids, and chemosed conjunctiva overlapping cornea. Vision was reduced to an obscure perception of light over both lids, all round the eyeball, but especially over the inner part of the upper eyelid; there was a strong pulsation synchronous with the cardiac beats and arrested by compression of the left carotid. Two days later eye more prominent; pulsation increased; loss of power to distinguish light from darkness.

Treatment and Result.

Sixteen days after accident ligature of left common carotid; pulsation ceased, swelling subsided, and power of moving eyeball returned; vision gradually improved and became nearly normal. Power over external rectus not regained till after she had left the hospital.

21. *Date, Surgeon, and Reference.*

1863. Holmes, E. L., M.D., Surgeon to the Chicago Charitable Eye and Ear Infirmary. 'Amer. Jour. of Med. Sci.,' July, 1864. "Aneurismal Tumour of the Orbit; Recovery."

Sex, Age, Side affected, Origin.

M. 23. Right eye. "Received on the *left* side a charge of No. 5 shot from an ordinary fowling-piece; some of the shot entered the left lung and the left kidney, several struck the side of the head, and two at least entered the left eye."

Symptoms and Progress.

Vision at once destroyed. Severe renal and pulmonary symptoms followed, but subsided, and the patient resumed his duties. At this time he noticed a slight protrusion of the *right* eye, conjunctival œdema at the lower part, and a peculiar blowing sound in the right side of the head. An attack of bilious fever aggravated the symptoms. Six weeks after the following

were the appearances of the left eye:—Complete ptosis; congested conjunctiva; iris discoloured; pupil contracted, filled with lymph, and drawn upwards to the sclerotic; globe slightly atrophied, and only painful on pressure; fistulous opening in sclerotic. *Right* eye so far protruded as to prevent closure of lids; chemosed conjunctiva; a fold concealing the lower lid; upper lid slightly œdematous, and its motions impaired; iris and pupil normal; vision perfect; tendency to photophobia; pulsation not to be seen on inspection, but revealed by moderate pressure with the fingers on the upper lid, and then it became evident to the sight; loud souffle over eye and temple, described by patient and physicians as like the puffing of a high-pressure engine heard at a distance; pressure on carotid rendered pulsation scarcely perceptible; pulse 116.

Treatment and Result.

Superficial scarification of the conjunctiva of the right eye produced accidental hæmorrhage to the extent of a pint without benefit or injury. Five drops of Veratrum viride every three hours, and Tilden's fluid extract of ergot four times a day. Nausea was controlled by diminishing the dose of Veratrum. Pulse fell to 80, then to 40. No change for a fortnight, except the presence of a second sound like the sharp whistle sometimes heard in the smaller bronchi. At the end of second week exophthalmos and chemosis began to subside, and souffle was subdued. All symptoms gradually improved, and at the end of six weeks patient went home and continued treatment for three weeks. Subsequently the eye became normal.

22. *Date, Surgeon, and Reference.*

1863. Legouest. 'Mémoires de l'Académie Impériale de Médecine,' t. xxvii, 1865-66, p. 156. "Aneurysme Traumatique de l'Artère Ophthalmique Gauche; insuccès de la compression indirecte; ligature du tronc carotidien et de la carotide externe; guérison."

Sex, Age, Side affected, Origin.

M. 21. Left orbit. Was carrying a large beam on his shoulders. Some companions threw the beam to the ground, thereby upsetting him, and he fell beneath it, and it struck the left side of his head and neck.

Symptoms and Progress.

Insensible for five hours. After recovery from effects of accident he went to the Hôpital Beaujon. At the end of seven weeks from the injury his condition was as follows:—Considerable exophthalmia; orbit filled with a tumour without precise limits; palpebral furrows obliterated; eyelids swollen, blue, and traversed by varicose veins; chemosis; paralysis of sixth nerve; very marked internal strabismus; sight of left eye disturbed, but preserved; diplopia; movements of upper eyelid sluggish; pulsation on laying fingers on globe and the tumours outside of it; over orbit a very strong "bruit de soufflet" like a forge bellows, continuous with reinforcements, synchronous with the pulse; bruit not heard on forehead, temple, or cheek; pressure on common carotid suspended bruit and pulsation, and diminished tension and vascularity of the eyelids and of the eye; pains round the orbit and temples, and in the ear, &c.

Treatment and Result.

Digital compression by medical students on five days for ten hours daily, with interruption of two hours for breakfast, three hours for dinner, and nine hours in the night for sleep. After forty hours' compression no real effect on tumour. Compression tried again with only six students for five days; after it had lasted nineteen hours it was abandoned. The disease increased; a voluminous chemosis covered almost entirely the ocular globe; inferior eyelid everted and covered with ulcers at seat of scarifications. Vision preserved, but troubled; parts menaced with inflammation; severe pain. Left common carotid tied; a slight bruit remained. External carotid tied; bruit ceased; all symptoms steadily disappeared. Five months later good but impaired vision; only internal strabismus left.

23. *Date, Surgeon, and Reference.*

1863. Nunneley. 'Med.-Chir. Trans.,' vol. xlviii, p. 17.

Sex, Age, Side affected, Origin.

M. 40. Left eye. Thrown from his horse while intoxicated.

Symptoms and Progress.

Insensible thirty-six hours. Free bleeding (more than Oss) in three or four days from left ear. Probable fracture of the base. In a few days lids swollen and red; chemosis; much pain; sight impaired; thought to be a case of conjunctivitis. In a month eyelids swollen, dark, incapable of closure; much chemosis of a purple colour; considerable proptosis and dilatation of superficial vessels; pupil dilated, inactive; lens muddy; vision materially impaired; distension and aching in globe; pulsation of globe perceptible to sight and touch; bruit and confusion in the head; great noise and pulsation in left ear. All symptoms increased on lying down, and relieved by pressure on the carotid; symptoms became aggravated and perception of light only remained.

Treatment and Result.

Ice, digitalis, rest, ineffectual. Five weeks after injury left common carotid ligatured; confusion; noise in head and ear instantly ceased, and hearing improved; protrusion and bruit lessened. Improvement, followed by relapse, and then a second subsidence. At the end of three months quite well; vision good. Quite well a year afterwards.

24. *Date, Surgeon, and Reference.*

1865. Nélaton. Published for the first time in Dr. Delens, op. cit. Abridged in Holmes' "Lectures," 'Lancet,' 1873.

Sex, Age, Side affected, Origin.

F. 17. Left eye. Fell from a voiture in July, 1864. Entered hospital February 25th, 1865.

Symptoms and Progress.

Did not lose consciousness. Blood flowed from the mouth, the nose, and both ears. Violent pains in head on left side; delirium for eight days; abscess in ear and left facial paralysis. Strabismus of left eye; swelling of

the conjunctiva; exophthalmia; aneurismal pulsations of the upper lid, and bellows murmur. Chemosis covering everted lower lid; bruit continuous with intermittent reinforcements; eye moveable; vision perfect; a small, rounded, moveable tumour, pulsating synchronously with the arterial beat without thrill, yielding to the pressure of the finger, about the size of a nut, and readily reducible, was felt at the upper and inner part of the left orbit. Besides continuous murmur and intermittent whiz, there was heard at irregular intervals a piping, whining sound ("bruit de piaulement"). Noise and pulsation perceptible, but not very inconvenient to patient. Compression of carotid stopped both and lessened tumour, but not exophthalmos. According to the patient exophthalmos immediately succeeded the accident. The chemosed conjunctival pad was not developed for five months. M. Nélaton diagnosed an arterio-venous communication in the cavernous sinus.

Treatment and Result.

Digital compression too irksome to patient; direct compression on globe; increased chemosis and congestion. Ligature of left common carotid about eight months after accident. March 6th, 1865.—Pulsations almost completely abolished; bruit remained; chemosis suppurated and diminished; exophthalmos lessened. Rigors, vomiting, on 11th and 13th. 17th.—Death. Post-mortem.—Fracture traversing body of sphenoid and petrous portion of temporal on each side. On left side a sharp point of temporal bone detached, and had probably penetrated the carotid and the wall of the sinus. Cavernous sinus and ophthalmic vein dilated and tortuous. Small hole in carotid below and externally communicating with the sinus.

25. *Date, Surgeon, and Reference.*

1865. Szokalski and Vrosinski. 'Annales d'Oculistique,' 1865, vol. i, p. 116. "Anévrysme Traumatique Diffus de l'Orbite."

Sex, Age, Side affected, Origin.

M. 50. Left orbit. Struck his left temple on two occasions whilst cutting wood.

Symptoms and Progress.

Some days after the left eye became a little more prominent, and then patient remarked there a flattened spot and fluctuating tumour, which a country barber wished to open. The patient consulted M. Szokalski. He had proptosis (one third of an inch); movements of globe upwards and outwards limited; sight preserved; fatiguing diplopia; a flattened tumour, extending deeply into the orbit, projecting in front, could be felt between the globe of the eye and the orbital arch; it presented a marked expansive pulsation. The left temple, strongly prominent, presented another tumour separated from the former by the external and superior border of the orbit; it had the extent of a five-franc piece, raised the temporal muscle, and extended in front as far as the circular temporo-frontal line. Pulsation in both tumours synchronous with the pulse, and pressure on the temporal tumour increased the tension of the tumour in the orbit. It was clear that

there were two communicating cavities filled with fluid. There was a bruit, and pressure on the carotid arrested both it and the pulsation in both tumours. M. Szokalski considered that the anastomoses between the lachrymal and temporal arteries had been torn by the injury.

Treatment and Result.

Digital compression for fifty-six hours at a time failed. Ligature being refused, the patient went out. Three months later he returned. Proptosis increased, and orbital tumour had passed the osseous border. Compression again failed. Left common carotid tied by M. Vrosinski; pulsation at once ceased; no cerebral disturbance; pains in orbit and forehead persisted for some time. After two months tumours softer and not pulsating. Some time later the tumours had shrunk considerably, and eye had partly returned into orbit. A small tumour about the size of a pigeon's egg over the left iliac crest had appeared, and was regarded as an aneurism of the circumflex iliac artery.

26. *Date, Surgeon, and Reference.*

1865. Erichsen. 'Science and Art of Surgery,' vol. ii, p. 88.

Sex, Age, Side affected, Origin.

M. 44. Left orbit. Fell on his head.

Symptoms and Progress.

Some months later eyeball greatly protruded; conjunctiva and eyelids congested and swollen. Bruit and pulsation very loud and distinct.

Treatment and Result.

Ligature advised by Mr. Erichsen and Mr. Greaves, of Boston, but declined. By attention to habits of life, abstinence from stimulants, &c., the tumour subsided and eye retreated. At the end of fourteen months the symptoms had, to a great extent, disappeared.

27. *Date, Surgeon, and Reference.*

1866. Desormeaux. Thèse de Laburthe, 1867. 'Des Varices Artérielles et des Tumeurs Cirsoides; de leur traitement specialement par les injections de perchlorure de fer.' Delens. Op. cit.

Sex, Age, Side affected, Origin.

M. 33. Left eye. Had his head jambed between a heavily-laden chest and a crane, February 2nd, 1866.

Symptoms and Progress.

Blood flowed from mouth, right nostril, and right ear. Sight at once lost in both eyes, and he even stated that both eyes were driven from orbits and hung over the cheeks. Right eye was soon completely lost; right upper jaw was broken and consolidated; hearing lost in right ear; right facial paralysis and anæsthesia. Patient heard a *bruit*, and believed there was a steam-engine at work under the ward, and only recognised his error when he found the noise follow him as a convalescent to Vincennes. About three months after accident the left eye was already prominent, but vision remained, and the patient could read nearly at normal distance; a tumour,

of the size of a small pea situated at the inner angle of the left orbit, appeared, a large vein proceeding from it and mounting up to the top of the forehead. The symptoms increased, and a year after the injury the left eye was markedly prominent; conjunctiva injected; cornea sound; pupil moderately dilated. Abduction only incomplete; partial ptosis; chemosed pad of conjunctiva overlapping lower lid; patient could read. Tumour of size of small nut, round, depressible, and not affecting the colour of the skin, yielding pulsations synchronous with pulse and evident both to the eye and finger. Two smaller pulsating tumours near it; veins in neighbourhood and on forehead enlarged. A large vessel ran vertically along the forehead from the eyebrow to the root of the hair, continuous with the larger tumour, nearly as large as the little finger, and slightly flexuous, and having the aspect of a varicose dilatation; its walls seemed thickened and pulsated vigorously; near the eyebrow it was constricted. M. Desormeaux thought it was the trunk of the ophthalmic artery, and diagnosed a cirsoid aneurism of that vessel. Others thought it to be the dilated vein. Strong pulsation of globe, raising finger laid on it; very intense *bruit de souffle* continuous with redoublements during arterial pulse, and at variable intervals the *bruit de piaulement*, which was very intense on the forehead. Pressure on carotid arrested bruit and pulsations, &c., but was not well borne by patient. Noise in head only heard at intervals by patient, and louder in recumbent posture.

Treatment and Result.

Rather more than a year after accident an injection of eight drops of perchloride of iron was made into the dilated vessel in the frontal region, and caused cessation of the pulsations in it. During the injection local pressure was kept up on the surrounding vessels. Three weeks later a second injection of twelve drops. Two months afterwards (13th May, 1867) the patient left the hospital; the tumour had much diminished, and there was neither pulsation nor bruit. Sight nearly normal; a little induration at the level of the tumour. M. Wecker found with ophthalmoscope a pale papilla, indications of hypertrophy of cellular tissue, very dilated veins, and some little hæmorrhagic spots.

28. *Date, Surgeon, and Reference.*

1866. Collard (de Berne). 'Gazette Médicale,' 1866, p. 631. "Anévrysme Traumatique de l'Orbite Gauche."

Sex, Age, Side affected, Origin.

M. 41. Left eye. Fell on to the back of his head.

Symptoms and Progress.

Insensible; small wound on left side of occiput; no signs of fracture. Eight or nine months afterwards a tumour had developed at inner angle of left eye, of thickness of a haricot, pulsating with vibration; hissing noise in ear; eyes engorged with blood and very prominent, left more than right; very marked congestion of lids; diplopia. *Diagnosis.*—Dilatation of ophthalmic artery and branches arising from and maintained by a morbid

state of the ophthalmic ganglion which furnishes vaso-motor filaments to the arteries of the eye.

Treatment and Result.

After some time a course of purgatives, with calomel and belladonna pill daily, and cold lotions to the forehead. Then tonic treatment was pursued. Exerting himself unduly when he was improving, he suffered a relapse. The symptoms were again subdued by leeches, calomel, belladonna, and cold lotions. Two years and ten months after accident he got some light work, and at the end of two months there was sensible improvement. In two or three months more the eye had re-entered the orbit, the tumour and dyplopia had gone, and he was cured.

29. *Date, Surgeon, and Reference.*

1867. Laurence, Zachariah, Surgeon to the Southwark Ophthalmic Hospital. 'Brit. Med. Journ.,' October 5th, 1867, vol. ii, p. 289. "A Case of Traumatic Aneurism of the Orbit in which the Common Carotid Artery was successfully tied."

Sex, Age, Side affected, Origin.

M. 41. Left orbit. He was an habitual drunkard, and on Monday January 14th, 1867, after a drinking bout, was taken home intoxicated at 3 a.m. During his staggering journey he fell down several times on to the kerbstone on to the back of his head, where there was a small wound. On reaching home he fell down in the passage, and there his wife let him lie.

Symptoms and Progress.

At this time he vomited considerably, and his wife noticed something amiss with his left eye. On recovering from his stupor three hours afterwards he complained of an aching pain on the left side of his forehead; slight proptosis of left eye. Then he had a fit with convulsions and stertorous breathing. Vomiting continued on and off for a week. Eight days after accident great proptosis; complete ptosis; immobility of globe; pupil fixed; perception of light; great chemosis of conjunctiva. Four days later Mr. Laurence observed pulsation of the eyeball, ceasing on pressure on the carotid; a distinct bellows murmur audible over the left side of the head. Patient heard a blowing bellows sound in left temple; upper lid livid and œdematous; details of fundus showed no special features; violent pulsation of carotid without bruit; slight epistaxis nearly every day.

Diagnosis.—"Rupture of ophthalmic artery near its origin from carotid consequent on fracture of the base of the skull."

Treatment and Result.

Compression with Skey's tourniquet applied for twelve days almost constantly. The effects were cessation of pulsation and bruit. The eyelids became more flaccid, paler, and cooler. The action of compression was aided by the local application of ice and ten-drop doses of tincture of digitalis and tincture of opium; no permanent benefit resulted. On February 19th, less than four weeks after the accident, the left common carotid was tied; pulsation ceased, and proptosis was lessened. April 23rd.

—Extreme ptosis; no congestion of eyeball, which was recovering its movements; cornea misty; still a line of chemosis. June 26th.—Eyeball in orbit; ptosis nearly gone; movements nearly perfect; vision lost.

30. *Date, Surgeon, and Reference.*

1867. Bell, Joseph, Lecturer on Surgery, Edinburgh. 'Ed. Med. Journ.,' July, 1867, p. 36. "Case of Pulsating Tumour of the Orbit cured by Ligature of the Common Carotid Artery."

Sex, Age, Side affected, Origin.

M. 42. Left orbit. Knocked down by an omnibus thirteen months before being seen by Mr. Bell. Lower jaw fractured, and head cut.

Symptoms and Progress.

Deep scar on left eyebrow. Since accident occasional paroxysms of intense headache; confusion and noise in head. A month after accident protrusion of left eyeball began, and steadily increased. Six months afterwards his sight began to fail rapidly. When seen by Mr. Bell he had extreme proptosis; both eyelids stretched and everted, especially the lower one; chemosed pad of conjunctiva; exposed cornea dim and lustreless. A tumour existed in the orbit, pulsating, compressible, and very soft. One specially rounded tumour just above the ball projected in the shape and size of a filbert, and was very soft. The supra-orbital artery and vein were both much enlarged and tortuous; the artery nearly as large as the radial, pulsated feebly. Well-marked bruit distressingly audible to the patient was heard over the head and neck. Pulsation and bruit ceased on compressing the carotid.

Treatment and Result.

Being almost blind with the right eye owing to a corneal nebula he was exceedingly anxious for operation. April 15th.—Left carotid tied; three hours after operation pulsation almost gone. In three days pulsation gone entirely. Cured with fair vision.

31. *Date, Surgeon, and References.*

1867. Foote, Dr. Case reported by Dr. Williams, of Cincinnati. 'New York Medical Record,' April 15th, 1868, vol. iii, No. 52, p. 75. Abstract (here given), by Dr. Noyes, 'New York Med. Jour.,' 1869, 665.

Sex, Age, Side affected, Origin.

M. 20. Left eye. Had a blow and depression of skull two and a half inches long, from vertex to left frontal boss, seven months before entering the hospital, June 15th, 1867.

Symptoms and Progress.

Immediately afterwards the eyeball protruded. When examined the external vessels of the left eye were very much increased in number and size; pulsation and thrill very strong. By ophthalmoscope the retinal vessels seemed much enlarged and tortuous, the optic nerve swollen, the borders ill-defined, grey in colour, and speckled by minute ecchymoses; along the veins some extravasations were seen, and the whole appearance was that of neuro-retinitis.

Treatment and Result.

June 22nd.—Carotid tied. Thrill and murmurs ceased, but returned in two hours; vision, which consisted in ability to count fingers at two feet, unaffected. After thirty days, the symptoms not being relieved, the other carotid was tied. Bruit and thrill silenced, but returned in five minutes; retina clearing. Three weeks after second operation vision improved; bruit very faint. August 21st.—Discharged cured.

32. *Date, Surgeon, and References.*

1869. Schiess Gemuseus and Socin. 'Klin. Monat. für Augenhlk., viii, p. 57, 1870. Shortly noticed in 'Ann. d'Oculistique,' 1871, vol. lxvi, p. 253. "Aneurism of the Orbit and Exophthalmos." Abstract, 'Oph. Hosp. Rep.,' vol. viii.

Sex, Age, Side affected, Origin.

F. 40. Left eye. In December, 1867, she received a blow from a horse's foot on the left side of the face while intoxicated. She was found insensible on the ground the next morning, her face greatly swollen, and she was bleeding from a wound under the eye.

Symptoms and Progress.

Unable to open eye for three months, and then she kept it closed because she saw double. From a very early period she heard a rushing or sawing sound in the left temple. Protrusion did not increase much after the first few months. In April, 1869, the symptoms were—exophthalmos; profuse lachrymation; congested conjunctiva; neighbouring veins congested; upper lid swollen and projecting; tumour above the eye compressible, fluctuating, very elastic, spread outwards under the orbital wall as far as the lachrymal region, inwards to the frontal vein, and upwards over the orbital margin, as a spindle-shaped swelling. Eyeball movable by patient. No ptosis. Pulsation and thrill in every part of tumour visible in profile. Pulsation of eyeball. Loud systolic bellows murmur, and shorter diastolic bruit. Compression of carotid stopped bruit and pulsation, and made tumour flaccid. Eyeball could then be pressed further back into orbit. Diplopia, the left image being the lower of the two. Vision with left eye $= \frac{1}{2}$. Congestion, prominence, and want of definition, of the optic disc. *Diagnosis.*—Aneurism of the orbit, probably of the frontal artery. Thrill and bellows murmur on carotid. In three weeks tumour larger.

Treatment and Result.

Three injections of ergotine subcutaneously caused vomiting, œdema, and increase of tumour. Digital compression for eight days, for from one to five hours daily, and then at the end of another week renewed for a week. No improvement. Prof. Socin tied the carotid, June 15th, 1869. All symptoms at once subsided, but half an hour later there was slight pulsation in sac. At the end of five weeks pulsation, bruit, and thrill had returned. At the end of five months the state of matters was much the same as before the operation. An attempt was made to ligature the frontal artery, but a network of vessels was encountered. Several were tied. June 29th, 1870.—The

tumour pulsated feebly, and was very compressible. Compression of right carotid stopped bruit and pulsation.

33. *Date, Surgeon, and References.*

1869. Harlan, George C., M.D. "Case of Traumatic Aneurism of the Orbit treated by Compression." 'Amer. Jour. Med. Sci.,' July, 1870. Also abstract and engraving in Dr. Morton's paper in the same number of journal.

Sex, Age, Side affected, Origin.

M. 25. Both eyes. While standing on the platform of a car in motion received a severe blow on the head from a car moving in the opposite direction. Lower jaw fractured on both sides and severe scalp wounds were inflicted. This was on 20th June, 1868.

Symptoms and Progress.

Partially insensible for three weeks. On recovering consciousness right eye very prominent and swollen, and he heard a roaring sound like water in his eye with every stroke of the pulse. A "red tumour" appeared on right eye and interfered with vision; it was cut off by medical attendant. A scar was left on conjunctiva, showing that the tumour was a chemosis. In two months the left eye began to protrude. During most of this time he had been at work, suffering inconvenience only from the deformity and noise which he referred positively to the right eye. August 14th, 1869.—Great exophthalmos of both eyes, greater of left, which was almost dislocated. Left ball could be pressed back, but not the right. Pupils freely movable; vision good. A loud aneurismal murmur audible over any part of the head and face. Pulsation of right eyeball. Bruit and noise stopped by pressure on right carotid; no effect on either eye by compressing the left carotid; no effect on left eye by compressing either artery. The right globe could be pressed back during compression of right carotid.

Treatment and Result.

Tinct. Veratri Viridis given as an adjunct to digital compression. Pulse reduced from nearly 100 to 44 and 56 beats a minute. Digital compression for eight hours first day, and then chiefly by patient for from four to six hours daily for five weeks. Sometimes a tourniquet, sometimes a stick with a pad, was substituted for patient's thumb. Subsequently relays of students kept up pressure uninterruptedly for twenty-four hours. Effects alteration of tone in bruit and noise in head, and decided diminution of exophthalmos of left eye. He returned to his occupation in a few weeks as bad as ever.

34. *Date, Surgeon, and Reference.*

1869. Wecker. 'Annales d'Oculistique,' 1869, t. i, p. 189. "Sur les Tumeurs Pulsatiles de l'Orbite."

Sex, Age, Side affected, Origin.

M. 31. Right eye. Fall from a ladder and wound over superior orbital border.

Symptoms and Progress.

Insensible for a quarter of an hour; wound healed, and he returned to work at the end of three months; considerable proptosis; chemosed pad of

conjunctiva. Movements of globe impeded; adduction abolished; distension of vessels at level of internal palpebral ligament, and there only a strong thrill synchronous with cardiac systole; intermittent bruit. Pressure on carotid stopped pulsation and bruit. Full account of ophthalmoscopic examination.

Treatment and Result.

Compression, application of ice, and mild purgatives. Some diminution of swelling, and slight improvement in vision. The patient left Paris.

35. *Date, Surgeon, and Reference.*

1869. Morton, Dr. T. G. 'Amer. Journ. Med. Sci.,' July, 1870, p. 43. Case 2, with engraving.

Sex, Age, Side affected, Cause.

M. 25. Left eye. Blow with the end of a loaded whip directly over the left eye; considerable swelling and ecchymosis.

Symptoms and Progress.

Twelve months later a slight swelling or lump appeared which he could grasp with his finger, soft and compressible; gradual slow growth, displacing eye downwards and outwards. At the end of two years it advanced more rapidly. For a long time he experienced a hissing or purring noise, more at night and in stooping posture. Vision gradually diminished after development of tumours. At the end of four years symptoms were—strongly marked exophthalmos; tumour above the eye extending backwards, soft, compressible; with some effort globe could be forced partly within the orbit, the tumour diminishing, and with the removal of the pressure the growth slowly regained its size; slight pulsation with feeble bruit; pressure on carotid did not entirely control the whir; pupil active; retinal veins full and dilated; disk atrophied; irregular fundus.

Treatment and Result.

The diagnosis being rupture of orbital vessels, followed by absorption of effused blood, and subsequent dilatation of the vessels arterial and venous, which were ruptured so as to produce an aneurism by anastomosis, Dr. Morton was of opinion that a partial removal of the tumour with the knife and ligation of the deeper portion would probably be sufficient.

36. *Date, Surgeon, and Reference.*

1869. Lawson, George. 'Brit. Med. Journal,' Dec. 11, 1869, p. 631, "Diffuse Orbital Aneurism in a Boy."

Sex, Age, Side affected, Cause.

M. 15. Left orbit. Five years before entering Middlesex Hospital he was jumping off a wall four or five feet high, when he fell on the end of a stick, which struck the inner side of the left orbit and pushed the eye outwards.

Symptoms and Progress.

Considerable bleeding, he thought "about a pint," a week after the accident; the eye was as prominent as on admission. Symptoms.—Exophthalmos to about half an inch; movements of eye in different directions in no

wise impeded; tortuosity and dilatation of conjunctival vessels; lids easily closed over globe; no tumour perceptible either to sight or touch; no visible pulsation of the eye; no pulsation when fingers were placed firmly against the globe; a distinct bruit audible over the left temple and forehead, and even slightly on the opposite side of the head, increased after exertion. Pressure on the carotid at once arrested the "slurring sound," which he compared to the blowing off of steam from an engine in motion, and controlled the bruit so completely that he frequentiy pressed the vessel with his finger to stop the noise in his head.

Treatment and Result.

Digital compression could not be maintained by the lad for more than two minutes at a time on account of the giddiness and faintness which it produced, whilst the pain rendered it intolerable. The effect of compression led Mr. Lawson to consider it inexpedient to ligature the carotid.

37. *Date, Surgeon, and References.*

1871. Galezowski. 'Gazette des Hôpitaux,' 1871, pp. 237, 241, 245, "Sur l'Exophthalmie consécutive à une Tumeur Vasculaire de l'Orbite." "Maladies des Yeux," 'Annales d'Oculistique,' 1871, t. lxvi, p. 104.

Sex, Age, Side affected, Cause.

F. 42. Left eye. Three years and a half previously she fell in the street and was wounded in the border of the orbit of the left eye. The eyelids continued red for fifteen days; since that time she had suffered nothing.

Symptoms and Progress.

Six weeks before seeing M. Galezowski, on rising in the morning, she had a headache, with pain and nausea, lasting the whole day. The headache resembled that to which she had been subject since seventeen, at the period of the courses. Whilst lying down she vomited bile, and afterwards heard a noise like that produced in sawing wood. In two days the eye was closed. When seen the eye was greatly projected and immovable; ptosis; pupil a little irregular; sight good; chemosed pad of conjunctiva covering lower lid; pains at root of nose and hemicrania; no engorgement of vessels of eyelids; retinal vessels engorged; double bruit de souffle and bruit de piaulement; feeling as if the eye was being pushed out of the orbit; no mention of pulsation or pulsatile tumour. Case seen also by MM. Gosselin, Richet, Labbe, Delens, and Gueneau de Mussy, and considered to be arterio-venous aneurism and exactly like M. Nélaton's case.

Treatment and Result.

Compression of the eye by bandage, iodide of potassium, and digitaline for nightly pains; pains ceased; compression continued for a month. Digital compression of carotid for fifteen or twenty minutes every two or three days, then increased to forty-five or sixty minutes daily; marked relief after each sitting. In a month movements of eye and eyelid had returned; chemosis gone. In the month of April the treatment was interrupted for a fortnight, and then continued every two or three days till July. In August the protrusion had almost entirely disappeared; no bruit audible either to patient

or surgeon. Treatment was being continued with every prospect of cure at time of publication of the account.

38. *Date, Surgeon, and References.*

1873. Hippel, Dr. A. V., and Schonborn. Prof. Graefe's 'Archiv f. Ophth.,' Bd. xx, Abth. 1, 1874, "A Case of Retro-ocular Aneurism, with Extreme Prominence of the Right Eye, after Injury to the Head." Abstract in 'Ophth. Hosp. Rep.,' vol. viii.

Sex, Age, Side affected, Cause.

M. 21. Right eye. Six weeks before coming under Dr. Hippel he had fallen from a horse, and, his foot catching in the stirrup, he was dragged along for some distance; he struck the left side of his head.

Symptoms and Progress.

Picked up insensible and bleeding from the nose, mouth, and left ear; both eyes bloodshot, and left pupil very small compared with right. In a few hours prominence of left eye; this disappeared; the right became congested and in a few days projected. For three weeks patient was insensible; on recovering he could scarcely open right eye or move eyeball; repeated attacks of epistaxis. Exophthalmos increased without pain; loud humming noise in left ear, the hearing in which had been defective since the age of four. When seen, left eye normal; commencing ulceration of right cornea, with extreme congestion and great chemosis; very small pupil, sensible to light; retinal vessels dilated and tortuous; no pulsation or tumour; loud bruit, hard-blowing and systolic, audible all over the head; bruit and noise ceased at once on compression of carotid. Diagnosis.—Diffused aneurism, either of internal carotid in cavernous sinus or of ophthalmic artery. The absence of pulsation and tumour of orbit contra-indicated an aneurism of that cavity. On review subsequently to ligature Dr. Hippel considered it a case of arterio-venous communication between the right internal carotid and cavernous sinus.

Treatment and Result.

Compression by bandage; diminished prominence of eye. Digital compression for five days, during several hours daily diminished bruit for a short time. Right common carotid tied by Prof. Schonborn rather more than three weeks after admission, on antiseptic plan; the bruit stopped at once, but returned feebly in three quarters of an hour. Compression of left carotid, tried several times, had no permanent effect upon the bruit; exophthalmos and congestion diminished; sight improved so that patient could read the smallest print, and cornea healed. In two months there was still the bruit, with some projection of globe and congestion. Compression of left carotid was tried for two or three hours daily for several days, but without benefit.

39. *Date, Surgeon, and Reference.*

1874. Rivington, W., Surgeon to the London Hospital. 'Med.-Chir. Trans.,' 1875.

Sex, Age, Side affected, Cause.

M. 24. Left eye. Blow on right side of head from iron step of an engine.

Symptoms and Progress.

Fracture of base of skull. Symptoms developed at the end of seven weeks.

Treatment and Result.

Digital compression on several occasions; mechanical and direct compression; Tinct. Ver. Vir.; injection of perchloride. Ligature four days after injection; cure with some opacity of cornea and persistence of slight bruit.

40. *Date, Surgeon, and Reference.*

1874. Nieden, Dr., of Buchom. 'Klin. Monatsbl. f. Augenheilkunde,' Januar Heft, 1875.

Sex, Age, Side affected, Cause.

M. 19. Left eye. Nearly six months previously to being seen by Dr. Nieden a large mass of coal had fallen on his head, causing slight abrasion of the scalp.

Symptoms and Progress.

Insensible till the next day; violent pain in the head, giddiness and nausea, and some bleeding from the nose. Twenty-four hours after the accident loud buzzing in left side of head with palpitation of the heart; prominence and redness of left eyeball. Next day proptosis as marked as at any subsequent time. Soon able to get about, the proptosis and noise in head remaining. After five months several attacks of giddiness and temporary loss of consciousness. Other appearances noticed by Dr. Nieden.—Upper lid much bulged forwards and dark red; movements limited; slight conjunctival chemosis; conjunctiva injected; the subconjunctival veins full, the injection disappearing on pressure; media clear; veins of retina turgid and tortuous; vision normal; diplopia; hearing on left side diminished; about half of eyeball may be replaced by light pressure, but there is then pulsation, which communicates a visible heaving movement to the hand, and the noise in the head becomes deeper and more intense; distinct thrill with each pulsation, and a prolonged blowing murmur synchronous with the ventricular contractions. All these phenomena disappear when the carotid is compressed. Diagnosis.—Diffuse retro-bulbar aneurism.

Treatment and Result.

Digital compression at first for half an hour twice daily, then by patient and nurses for eight or ten hours daily; patient's pressure effective. Elastic ring compressor used for four weeks. At the end of ten weeks, compression having been in vain, ligature of left common carotid on Lister's plan. Thrill, murmur, and pulsation ceased; noise in head greatly lessened; a distant blowing murmur could still be heard. Wound healed without complication six weeks after operation. Discharged with slight prominence of eyeball; very little injection of conjunctiva; about one fifth as much noise in head.

41. *Date, Surgeon, and Referenee.*

1874. Lansdown, F. P., Senior Surgeon to the Bristol General Hospital.

" A Case of Varicose Aneurism of the Left Orbit cured by Ligature of the Diseased Vessels," 'Brit. Med. Journ.,' June 5th, 1875, p. 736, June 12th, p. 771, and June 26th, p. 846.

Sex, Age, Side affected, Cause.

M. Age not stated. Left. Wound on inner side of left upper lid, caused by the bursting of a soda-water bottle. The wound penetrated the upper lid and was about half an inch in length. The eyelids became distended to their utmost by effusion of blood, and an artery was jetting blood from the wound. The edges of the wound were brought together by suture, and in a few days the patient was apparently well.

Symptoms and Progress.

In about six weeks the eye gradually became prominent, the eyelids swollen, and the veins of the conjunctiva tortuous and distended. In a few weeks a small pulsating tumour was discerned at the inner angle of the orbit, beneath the cicatrix of the old wound; there was a distinct murmur over this as well as over the eye. The sight was perfect. The exophthalmos was about half an inch in extent. The bruit was continuous, and heard only over the inner angle of the orbit. Engorgement of retinal veins. Bruit arrested by pressure on carotid.

Treatment and Result.

Rest and remedies to restrain the circulation. Pressure on aneurismal swelling applied by means of a truss gave much pain, and pressed the swelling into the orbit. Ligature of carotid advised by Mr. Bader and Mr. Higgins, of Guy's Hospital. On February 18th, 1874, Mr. Lansdown cut down and opened the old wound, exposing a small, white, glistening, globular, pulsating tumour, about as large as a good-sized pea, with a large tortuous vein coming off from the front of the sac and passing to the back of the orbit. Between the eye and the aneurism there were several smaller vessels going to and surrounding the sac, which made the operation tedious. Having found the feeding vessel Mr. Lansdown placed a carbolised ligature on the cardiac and distal sides of the aneurism and closed the wound. On the fourth day the sac was discharged from the wound. In a week the eye had returned nearly to its natural level, and the patient made a complete recovery. A year after the operation no one could notice any difference in his eyes.

OBSERVATIONS

UPON THE

ELIMINATION OF UREA IN CERTAIN DISEASES.

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I HAVE the honour of submitting to the Society some observations which I have lately made upon the excretion of urea in certain diseases.

Before doing so I wish to take this opportunity at the outset of expressing my thanks to Dr. Andrew for the kindness with which he placed all the advantages of his ward in St. Bartholomew's Hospital at my disposal, and of acknowledging how much I owe to his encouragement and assistance during the preparation of this paper.

The experiments were made by a method and apparatus described in a paper read by Dr. Russell and myself at one of the meetings of the Chemical Society about three months since, and published in the August number of the Chemical

Society's 'Journal.' I may refer for a full description of the method and apparatus to that paper, and I will pass without further comment to the observations I have made.

The following are the cases in which I have examined the excretion of urea:—Two cases of Addison's disease; three cases of pneumonia; four cases of rheumatic fever; and one case of diabetes mellitus.

I propose in each of these cases to give first the results of the experiments, then to make a few remarks on each case, and to complete my paper by drawing a few general conclusions which a review of the cases on a whole seems to suggest.

Each case is prefaced by a table giving the observations made each day during the period the case was under notice.

I will commence with the cases of PNEUMONIA, and I propose to divide the results into two groups, as I have also done, where it was possible, in the cases which are to follow, the first group containing the results *during fever*, and the second those *during convalescence*.

CASE 1.—The first case of pneumonia is that of Ellen C— (see Table I), and the following observations were made during a relapse:

TABLE I.—Ellen C—. Mary Ward (26). Pneumonia (relapse.)

Date.	Total urine.	P. c. urea.	Total urea.	Temperature.	Specific gravity.	Diet.
May 29th	385	3·4	13·10	—	—	May 26th.—D.L.[1] Milk Oj. Eggs ij. Brandy ʒij. Beef-tea Oj. Essence ʒv. Wine ʒiv, instead of brandy.
„ 30th	425	3·5	14·87	104·2	1030†	
June 1st	510	4·1†	20·91	104·3	1025.	
„ 2nd	375	3·7	13·88	103·8	1020.	

[1] D. L. = Dieta lactis. D. D. = Dieta dimidua. D. C. = Dieta carnis, of the St. Barthol. Hosp. Pharmacopœia.

Date.	Total urine.	P. c. urea.	Total urea.	Tempe-rature.	Specific gravity.	Diet.
June 3rd.						
„ 4th .	350 ...	3·3 ...	11·55.			
„ 5th .	600 ...	3·2 ...	19·2	... 102	... 1022.	
„ 6th .	500 ...	3·5 ...	17·5	... 104	... 1025	D. D.
„ 8th .	350 ...	3·6 ...	12·6	... —	... 1026	Eggs ij.
„ 9th .	350 ...	2·4 ...	8·4	... —	... 1018 †	Brandy ℥i ij.
„ 11th .	390 ...	3·5 ...	13·65	... 100	... 1026	Ale Oss.
„ 12th .	500 ...	3·4 ...	17·	... —	... 1022	Lemon j.
„ 13th .	275 ...	4·05† ...	11·14	... —	... 1032	
„ 15th .	250 ...	2·5 ...	6·25	... —	... 1022.	
„ 16th .	650 ...	2·2 ...	14·3	... —	... 1023.	
„ 17th .	675 ...	2·1 ...	14·18.			
„ 18th .	1150 ...	1·1 ...	12·65.			
„ 19th .	700 ...	2·1 ...	14·7.			
„ 20th .	800 ...	2·4 ...	7·2	... —	... 1026.	
„ 23rd .	— ...	— ...	—	... 99·3.		
„ 25th .	375 ...	2·65 ...	9·92.			

Smell very putrescent, with occasional traces of albumen, and a very deep colour with nitric acid.

During *the fever* (which lasted until the 15th June) we have these averages.

Total vol. of urine.		P. c. urea.		Total urea.
400 cc.	...	3·4	...	13·65.

Each column in Table I being taken separately, it will be seen that the total quantity of urine is much reduced, and, though fluctuating round the mean of 400 cc., cannot be shown at present to depend upon any definite cause. This reduction occurs also in spite of the extreme thirst of the patient. The percentage of urea remains fairly constant, varying from 2·4 to 4·1 and giving the mean of 3·4, with which most of the experiments agree. The two highest percentages, and it must be noticed they depart in a most marked way from the average, follow upon the alterations of diet. Crosses stand against the altered diet and the percentage in which it caused alteration. It must be noticed that these changes in diet produced only a temporary rise in the percentage, which falls the next day to the average, or

slightly below it. It would appear as though the balance of nutrition was disturbed by such changes in diet, and that it required some hours to restore itself. I do not know that it is possible to point out any but a very general relation between the percentage of urea and the temperature. As long as the temperature remains above normal the percentage is high. As soon as the temperature falls the percentage is reduced, but at present it cannot be stated that a rise or fall in temperature is attended by a rise or fall in urea percentage, nor can we show any inverse proportion between the urea percentage and the volume of urine.

In this case it is to the total excretion of urea in the twenty-four hours that I would wish to draw special attention. The mean excretion during the fever was 13·85 grammes, and the reduction in amount is very marked, and this, too, when the patient was upon an extremely rich nitrogenous diet, one which, until it was changed on June 12th, contained an amount of nitrogen to which that excreted in the form of urea bears but a very small proportion. I propose to return to this strange fact after speaking of the other cases of pneumonia.

I have to show lastly, in this case, the entire independence of the total excretion of urea upon the temperature, as is seen in the following table:

Temperature	.	. 104·2	...	104·3	...	104·.
Urea .	.	. 14·87	...	20·91	...	17·5.

During *convalescence* (which commenced on the 15th June) we notice, looking at the figures in the respective columns after the 15th June, a remarkable diminution from the normal in the volume of urine and the total excretion of urea. In the urea percentage are to be observed curious oscillations, to which I shall draw attention again, later. In this case the oscillations take the form of a gradual decrease, and again of a gradual rise.

It remains for me to add, before passing to the other cases of pneumonia, that the urine in this case contained occasionally slight traces of albumen, had a most unpleasant

putrescent odour, and gave an extremely deep reddish-purple colour with nitric acid.

Case 2.—The second case is that of Alfred E— (see Table II).

Table II.—Alfred E—, æt. 74. Mark Ward (20). Pneumonia (right side).

Date.	Total urine.	P. c. urea.	Total urea.	Tempe-rature.	Specific gravity.	Diet.
June 16th	—	—	—	—	1020	
„ 17th	600	3·	18·	102	—	D. L.
„ 18th	600	3·3	19·8	101·8.		Essence Oss.
„ 19th	700	3·6	25·2	101·9	1022	Arrowroot Oj.
„ 20th	925	3·2	29·6	99·9	1020	Egg j.
„ 21st	640	3·3	21·04	98·3.		Brandy ℥ss.
„ 22nd	640	3·3	21·04	101·7.		Beef-tea Oss.
„ 23rd	600	2·7	16·2	98·4	1020.	
„ 25th	900	2·	18·	98·4	1016.	
„ 26th	1200	1·85	22·2	—	1018	Chop.
„ 27th	1750	1·2	21·.			
„ 28th	1250	1·1	13·75.			
„ 29th	1550	1·05	16·3	98·2.		
„ 30th	1500	1·	15·.			
July 1st	1550	1·3	20·15.			
„ 2nd	1200	1·55	18·6.			
„ 3rd	1400?	1·65	23·1.			
„ 4th	1650	1·75	28·87.			
„ 5th	1425	1·75	24·94.			
„ 6th	1100	1·85	20·35.			
„ 7th	950	2·15	20·4.			
„ 9th	1150	2·3	26·45.			
„ 10th	700	2·2	15·4.			
„ 11th	?	2·2	?			

Went out of hospital well.

During *the fever* the mean results for the six days on which experiments were made are—

Total urine.	P. c. urea.	Total urea.
700	3·28	22·45.

The total volume is here considerably reduced, though not to the same extent as in the previous case. It is subject also to very considerable variations from day to day.

The percentage amount of urea is fairly constant, varying from 3·0 to 3·6, and giving as mean 3·28. Here again it is to be observed that the percentage does not vary inversely, or depend upon the volume in any way at present evident.

The total urea here again is certainly not above the normal, in spite of the increased temperature, though its reduction is not nearly so marked as in Ellen C—'s case. Variations from day to day are also to be observed which are independent of the urea percentage or of the temperature.

The total amount of urea is also considerably below what the quantity of nitrogen in the food would lead us to expect.

During *convalescence* the results show a gradual increase in the volume of the urine for the first few days, when it reaches its normal, though there are now, as during the fever, curious variations, for which we cannot account, in the total volume.

The percentage of urea which, during the fever, is nearly 3·3, falls gradually for the first seven days, until, on the seventh day, it is as low as 1·0, and it subsequently rises again slowly until it reaches a mean of about 2·2, at which it appears to become fairly constant.

A series of percentages such as these shows very clearly that there is at any rate no simple relation between the volume of the urine and the percentage amount of urea, to which fact, however, I shall have to refer again.

In the total amount of urea we notice, for the first week of convalescence, a considerable fall as compared with that excreted during the fever, so that the mean for the first seven days of convalescence is only 17·5 grammes. In the subsequent eight days a considerable rise occurs again, the mean being for these days twenty-three grammes, and in both these periods we have these curious variations from day to day.

Case 3.—The last case of pneumonia of which I wish to speak is that of G— (see Table III), in which the result was death. In both of the other cases recovery took place.

The results in this case are complicated by the nervous symptoms of the patient. The man had been a hard drinker, and died ultimately rather from his nervous symptoms than his pneumonia.

The temperature was high throughout, and the patient was in an extremely excitable condition, with tolerably frequent attacks of delirium.

The total quantity of urine varies considerably, giving as the mean 835 cc., a quantity larger than is found in either of the other cases. The same irregular variations from day to day are to be observed.

The percentage of urea gives as the mean 2·5, which is much lower than in the other cases, while the total urea excreted is subject to very considerable oscillations; is below the average, the mean being twenty-one grammes; does not depend in any way upon the temperature; and is below the amount we should expect from the food.

TABLE III.—G—. Mark Ward (9). Pneumonia.

Date.	Total urine.	P. c. urea.	Total urea.	Temperature.	Diet.
June 26th	700	2·2	15·4	103·	D. L.
„ 27th	950	2·	19·	103·9.	Beef-tea Oj.
„ 29th	Not saved	2·2	—	102·4.	Eggs ij.
July 1st	850	2·55	21·675	103·2.	Essence ʒv.
„ 3rd	1025	3·	30·75	101·1.	Brandy ʒvj.
„ 4th	650	2·8	18·2	102·2.	
„ 5th	Passed in bed.				
„ 6th	Ditto	2·8	104·.		

Edward P—. Mark Ward (20). Pneumonia.

June 13th	1250	2·15	26·875	—	Specific gravity 1015, acid with putrid smell.

R—. Mark Ward (15). Pleuro-pneumonia.

July 13th	600	4·2	25·2.		
„ 14th	250	2·85.			
	Whole not saved.				
„ 16th	500	2·225	11·125.		

In another fatal case (Edw. P—, in the same table) extremely like that just mentioned, in which also the nervous symptoms preponderated, and in which there was also a history of intemperance, we got, on the only day on which examination of the urine was possible, strongly analogous results. The percentage urea is much the same, though the total volume of urine, and the total amount of urea, are on this particular day much larger than the mean in the previous case.

It is interesting to observe that, while it is stated to be a physiological fact that nervous excitement increases the excretion of urea, though the nervous symptoms were the most prominent features in both these cases, the total excretion of urea is certainly not proportionately increased.

What general conclusions can be drawn from comparison of the cases already brought forward? There is a considerable reduction in the total volume of the urine, which agrees with the general statements found in books (Niemeyer, 'Medicine,' vol. i, p. 171; Parkes 'On Urine,' p. 270), though in different cases we find great differences. In the three cases given the averages are as follows:

Ellen C—.		Alfred E—.		G—.
400 cc.	...	700 c.c.	...	840 cc.

I cannot discover any very close relation between the volume of the urine and the temperature of the patient. The cause of this reduction is in great measure no doubt the increased perspiration, but the lungs doubtless play an important part in the diminution of water, while a third cause may possibly be the altered nutrition of the kidney which the common presence of albumen in the urine of pneumonic patients proves to exist (Parkes, 'Urine,' p. 270).

During convalescence the quantity of urine returns to the normal, as Table II shows (Parkes, 'Urine,' p. 270). *The percentage of urea* is in all cases very high, even in those in which there is a reduction instead of an increase in the total amount of urea.

In these cases the percentage is 3·4, 3·28.

In Moos (Henle and Pfeuffer, 'Zeitschrift,' n.f., iv, 3), 4·27.

In Vogel („ „ „), 4·8.

In Unruh (Virchow, 'Arch.,' 1869, 48, S. 227), 4·8, 4·3, 3·33, 3·62, 3·13, 4·57, 4·43.

I should state that there are two cases quoted by Vogel (*ut supra*) which give a very low percentage—1·25 and 1·5, and that in these cases the quantity of urine passed was also very large. In the two other cases mentioned above, viz., those of G— and P—, there is also a reduction in the percentage, viz., 2·15 and 2·5; but here we have complications on the side of the nervous system, so that it is hardly right to argue from these instances.

Though, as I have stated in analysing Case 1, there is no relation between the temperature and the percentage of urea from day to day, yet a very marked relation exists between the fall of temperature at the termination of the fever and the percentage of urea. With the fall in temperature a fall in the percentage occurs, which may be clearly seen in both the cases in this paper, and may be noted in experiments on pneumonia made by other observers (Unruh, *ut supra*).

As regards the total amount of urea we find very contradictory statements in the books. In all the cases I have cited above the amount of urea is certainly not above the normal, but, on the contrary, reduced, the reduction in the first case quoted being very remarkable, and still more so when the amount of nitrogen which the food contained is taken into consideration. In the first two cases we have a quantity of food consumed which contains at a very low estimate 280 grains of nitrogen, which is equivalent to 600 grains of urea, or 40 grammes, while Ellen C— excreted in urea only an average of 14·1 grammes, and E— of 23·0 grammes. The question arises at once, what can have become of this nitrogen? There are several possible means by which it could have escaped estimation.

1st. It may have been *not digested*, and so have never entered the system at all; but against such an explanation

are the facts that the appetite was good and that there was no diarrhœa.

2nd. If digested, it may have been *excreted or stored up*. If excreted, either it left the body by the kidneys, in which case it must have been in some other form than urea (let us suppose leucin, or tyrosin), or it left the body by some other channel, viz., the lungs, the skin, or the intestines. If stored up, the question remains, in what form, and where?

I am afraid I cannot do more than balance the probabilities of these various means of elimination. Experiment alone can decide the question, and at present we have none to guide us. It is improbable that the escape of nitrogenous products by the lungs should be sufficient to account for so great a deficiency. The skin is a more probable channel, but even this we could only conceive to account for part of the deficiency, and on the side of the intestines we have no evidence of such loss.

We are thus reduced to the two last possible means of elimination, and first by the kidneys in some other form. This is very probable; pneumonia being an affection of the chief oxygen-introducing organ in the body, we might expect to get deficient oxidation, and hence to find in the urine such substances as leucin and tyrosin, the primary products of the decomposition of albumen, which, under ordinary circumstances, are oxidised to urea (Schultzen and Riess, *ut supra*); or we might get uric acid, but I know of no experiments bearing on this question.

Lastly, it may be stored up.

(*a.*) By *retention*, in which case we should expect a critical discharge after the pyrexia, such as Dr. Parkes states to occur in such cases (p. 278); but in the case he quotes it was uric acid, and not urea, and this would, to a certain extent, support the theory of the excretion of nitrogen by the kidney in some other form than urea. However, in my cases, as regards urea no increase in its quantity was observed after the pyrexia.

(*b.*) By *going to form tissue*, and we might thus connect the absorption of nitrogen by the body with the increased

production of cells in the lungs and the disappearance of the chlorides; but it is unlikely that we should get such extensive carpentering without chips, and such great formation of new growth without proportionate waste, for in children, in whom cell-growth is very rapid, we have a proportionate increase in the production of urea. We should further expect that *resolution* would cause increase in the urea, and this we do not find. We have, moreover, direct statements to the contrary by Vogel and others, Moos, and Wachsmuth, who affirm that the excretion of urea is greater before than after resolution.

	Vol. of urine.		Urea.
On each of 3 days before resolution.	. 926 cc.	...	37·65
On each of 7 days during resolution	. 1073 cc.	...	21·16

If it be true that the absorption of nitrogen during the fever is due to cell-growth, the reason that such excess does not appear in the urine during convalescence may partly be that there is a subsequent reabsorption to reform the wasted tissues akin to that which occurs during violent exertion.

It seems, therefore, most probable that we have in pneumonia the elimination of nitrogen by the kidneys, but not in the form of urea. Whether this is so, and in what form it is excreted, remain for experiment to determine.

As regards the urea in convalescence from pneumonia, we notice, in both the cases quoted above, a diminution during the first period. In the latter stage it appears to be increased again and to reach about the normal amount.

	Ellen C—.		E—.
Convalescence, 1st period . .	. 12·16	...	17·0
During fever	. 14·1	...	23·0

From consideration of the cases of pneumonia in which analyses of the urea are given we are led to the conclusion that there must be two distinct classes of cases—1st, those in which the urea is increased, and 2nd, those in which it is diminished.

1. *Those in which it is increased.*

Dr. Parkes ('Urine,' p. 271) quotes a number of cases, reported by different observers, in which the urea ranged from 33·45 to 85·56 grammes, or from 516 to 1321 grains.

Vogel, Moos, and Wachsmuth, quoted in the same chapter by Dr. Parkes, state similar results.

Unruh (Virchow, 'Archiv,' 1867, 48, f. 227) gives a number of cases in which the average during the fever varied from 22·98 to 38·8 grammes.

Moos (Henle and Pfeuffer, 'Zeitschrift,' n. f. iv, 3), and Vogel („ „), make similar statements.

2. *Those in which it is diminished,*

as in the cases here quoted; in certain cases mentioned by Dr. Parkes as extremely rare; and in a few cases quoted by Unruh (*ut supra*), and in two quoted by Vogel (*ut supra*).

Dr. Parkes states that a small excretion of urea affords a bad prognosis; yet in the two cases I have quoted, in which there was marked deficiency, recovery took place; but even these bear out, in a fair degree, Dr. Parkes's statements, for, though in both the urea was reduced, yet in Ellen C—'s case, in which the reduction was most marked, convalescence was most tedious, and it is probable that recovery was not quite complete when she left the hospital, for her temperature never returned to normal, though all signs of lung mischief had disappeared, and we should expect, *à priori*, such marked reduction to be a bad sign, for it must mean serious interference with the normal nutrition of the body, if those substances which should leave it oxidised as urea are not so oxidised, and the prognosis will be better or worse according as there is less or more interference with the normal processes, of which in this respect urea may be taken as an estimate.

It remains only for me to add that the excretion of urea appears to vary very considerably in the different chest diseases. In pleurisy Dr. Parkes (p. 280) says it is almost natural. In bronchitis it is certainly not increased in

Brattler's cases ('Ein Beitrag zu Urologie,' 1858, p. 42), while Moos ('Zeitschrift f. rat. Med.,' B. vii, 362) gives a case which passed only nine grammes during the fever, while during convalescence twenty-four grammes was passed. It may be that these differences are to be explained by the different degrees to which oxidation is interfered with in the different cases. Unfortunately, in nearly all these cases quoted no reference is made to the diet of the patients.

I pass now to the cases of RHEUMATIC FEVER, of which the first is that of Thomas C—, (see Table IV), and the results in this case I propose to divide as before.

CASE 1. TABLE IV.—James C—, æt. 13. Mark Ward. Rheumatic fever.

Date.	Total urine.	P. c. urea.	Total urea.	Temperature.	Specific gravity.	Diet.
May 30th	860	2·85	24·51	—	—	Arrowroot made
„ 31st	1000	1·85	18·5	101	1015	with water.
June 1st	525	2·7	14·175	101	1015 frothy	Arrowroot biscuits, do.
„ 2nd	810	1·6	12·96	100	1010 just acid	Aquæ destil. ℥j ter die.
„ 3rd	1000	1·6	16·	—	1013 neutral.	Brandy ℥ij.
„ 4th	850	1·6	13·6	—	1012 neutral.	
„ 5th	750	1·2	9·	100·3	1012 neutral	June 6th.— Egg.
„ 7th	500	3·26	16·3	100	1020	„ 7th.—L.D.
„ 8th	500	4·34	21·7	—	1025 acid, deeper colour.	and fish.
„ 9th	400	3·8	15·2	99·6.		
„ 10th	830	2·8	23·24			
„ 11th	770	3·2	24·64	—	1023	
„ 12th	725	3·05	22·11	—	—	D.D.
„ 14th	950	3·	28·5	—	1023	Milk Oj.
„ 15th	1325?	2·35	31·14	—	1025.	
„ 16th	1075	1·55	16·7.			
„ 17th	1050	1·5	15·75.			
„ 18th	1050	1·5	15·75.			

Date.	Total urine.	P. c. urea.	Total urea.	Tempe-rature.	Specific gravity.	Diet.
June 19th	1375	1·75	24·06	—	1017.	
„ 20th	1150	1·75	15·52.			
„ 21st	925	2·05	18·96.			
„ 22nd	925	2·05	18·96.			
„ 25th	900	1·95	17·55.			

1st.—During *the fever* the averages are—

Total vol. of urine.	P. c. urea.	Total urea.
719 cc.	*	16·19.

The total volume of urea is much reduced, but shows very considerable variations, and it is to be observed that they are entirely independent of the temperature.

Temperature	100	100·3	100·.
Total vol.	810	750	500.

In discussing the results contained in the other two columns of the table it is impossible to separate the effects produced by the fever from those produced by the treatment. The treatment consisted in the absolute deprivation of nitrogenous food, so that the patient was placed upon a diet consisting of arrowroot made with water, arrowroot biscuits, and as medicine Aq. destil., ʒj ter die. This treatment, first practised by Dr. Andrew at St. Bartholomew's Hospital, and suggested to him by certain statements in Dr. Parkes's lectures, has yielded, in those cases which would bear it, extremely good results. They are described and discussed in the 'St. Bartholomew's Hospital Reports' for 1874.

The temperature in this case was not very high on the first day on which it was taken, but the patient had already been ill some days, and had been placed on a low diet before this treatment was adopted.

The patient began with this diet on the 29th of May. On the 30th the urea percentage is 2·85, and from that day progressively falls, the gradual fall being interrupted on the third day of treatment by one of those oscillations to which reference has been previously made. The fall is

extremely rapid, and on the fifth day the urea percentage is 1·2 only. The specific gravity follows fairly closely the fall in the percentage, and with it certain changes in the general character of the urine occur. Being at first of high specific gravity (1025) and strongly acid, its specific gravity falls until on June 5th it is 1012, and the acidity is reduced more and more until on the 3rd, 4th, and 5th the reaction is neutral. With this change in the percentage a similar change is observed in the total amount of urea. At first 24·5 grammes, a fairly large amount, it falls with a single oscillation (which, by the way, does not coincide with that observed in the percentage), until it reaches nine grammes, and it is possible that we have at this time eliminated the complication of food, and that we may take the nine grammes as the measure of the tissue waste.

In the remaining three days of the fever I propose to consider the effect of the change of diet upon both the percentage and the total amount of urea together. An egg was eaten on the 6th, and on the 7th we have a sudden rise in the percentage to 3·26 and an increase in the total amount of urea of about 7 grammes. The egg could not have accounted for more than 1·5 grammes on the most liberal allowance, so that we have a rise and increase totally out of proportion to the food. With the rise, it should be observed, there was a slight return of pains, which, while the percentage was low, had almost entirely disappeared.

On the 7th the diet was again changed, and again we notice a marked rise in the percentage and in the total amount of urea. On the third day of treatment both the percentage and the total urea fell, so that here we notice again those oscillations produced by change of diet to which I have before referred.

2nd.—During *convalescence* the averages are—

Total vol. of urine.		P. c. urea.		Total urea.
1000 cc.	...	*	...	21·16.

And we notice that there is a gradual rise in the total amount of urine, but with fluctuations from day to day such as all the other cases show.

The changes in the percentage of urea admit of the division of convalescence into two periods. In the first there is a gradual but progressive diminution in the percentage, and the second is characterised by a gradual and progressive rise, until a mean of about 2·0 is established. In the first period the average of total urea excreted is much higher than in the second, but we have here again variations from day to day for which we cannot at present account.

It is to be observed that the specific gravity here follows fairly closely the variations in the percentage.

Case 2. In the next case to which I now pass, that of William T— (see Table V), we have the fever at an earlier stage, and we notice here the small volume of the urine, the very high urea percentage (4·4 and 4·1) with at the same time a not excessive excretion of urea. As soon as the effect of the diet begins to make itself apparent we have a large increase in the total amount of the urine, a very considerable and sudden drop in the urea percentage, while, however, the total urea remains fairly constant, so that we do not get here, as in the last case, so marked an effect produced upon the total urea excreted. But this may be due to the fact that the patient was not kept so long upon a purely non-nitrogenous diet.

Table V.—Willam T—, æt. 16. Mark Ward (21). Rheumatic fever (pericarditis).

Date.	Total urine.	P. c. urea.	Total urea.	Temperature.	Specific gravity.		Diet.
July 2nd .	500 ...	4·4 ...	22· ...	101 ...	1025	Neutral.	Arrowroot biscuit, Arrowroot water, Aq. distil.
„ 3rd .	500 ...	4·1 ...	22·55 ...	99·3 ...	1025.		
„ 4th .	1100 ...	1·95 ...	21·45 ...	99·.			
„ 5th .	1200 ...	1·95 ...	23·4.				
„ 6th .	1100 ...	1·6 ...	17·6 ...	100·3 ...	—	Neutral.	Egg j. D. L.
„ 7th .	1150 ...	1·95 ...	22·4 ...	— ...	—	... — ...	Fish.

Date.	Total urine.	P. c. urea.	Total urea.	Temperature.	Specific gravity.	P. c. urea in ur. sang.	Diet.
July 8th	1150	2·	23·.				
„ 9th	750.	3·25	24·38.				
„ 10th	450	3·45	15·5	—	—	3·5.	
„ 11th	750 (?)	2·85.					
„ 13th	1000	2·	20·	—	—	2·6.	
„ 14th	750	2·5	18·75	—	—	1·8	July 15.—D. L. Milk.
„ 16th	1150	1·3	15·5	—	—	1·175.	
„ 17th	850	1·4	11·9	—	—	1·.	
„ 20th	—	—	—	—	—	0·9.	
„ 21st	—	—	—	—	—	1·225.	
„ 24th	1250	0·83	10·38	—	—	0·93.	

The effect upon the urea percentage and the total urea of change in diet is the same as in the previous case, except that the rise takes place a day later. Again, we notice in this case, as in the last, a diminution of the volume accompanying the rise in the percentage, though no definite relation can be traced between them.

During convalescence there is the same gradual fall in the urea percentage, but there is no subsequent rise again, probably because the observations were not carried so far into convalescence as in the previous case.

The averages during convalescence are—

Total vol. of urine.	Urea p. c.	Total urea.
830	*	17·7.

In the total amount of urea we observe here also a diminution in the amount excreted during the day.

Case 3. In the next case, that of Arthur S— (see Table VI), we have a series of facts which are suggestive as to the cause of rheumatic fever. This man was sent into Mark Ward for threatened rheumatic fever, but on examination the chief pain, which was in the hip, was found referable to a sprain which he had got while riding, and he was removed to a surgical ward. Here he was kept for a week on full meat diet and a pint of porter. His bowels did not act during the whole of this time, and he was sent back to Mark Ward,

complaining of considerable pain in his joints, and with every sign of commencing rheumatic fever, so that we have here a disease very similar to rheumatic fever produced by simple digestive derangement, and by accumulatiou in the body of effete nitrogenous products, which were too abundant for the kidneys to remove sufficiently. As soon as the supply was stopped in the food the surplus was quickly worked off by the kidneys and health restored.

TABLE VI.—Arthur S—. Mark Ward. Rheumatism (?).

Date.	Total urine.	P. c. urea.	Total urea.	Tempe-rature.	Specific gravity.	Diet.
June 23rd	—	4·1	—	—	—	Refused to take the food, and had none. Arrowroot water, arrowroot biscuits, Aq. distil.
„ 25th	650	4·45	28·9	98·5	1032	June 24th.—D. L.
„ 26th	1000	3·25	32·25	—	1025.	
„ 27th	650	2·5	16·25.			
„ 28th	1250	1·75	21·87.			

John E—, æt. 24. Radcliffe Ward. Rheumatic fever.

		P. c. urea in ur. sang.		Tempe-rature.		
June 11th	—	3·5	—	103·2	—	D. L. Milk Oj.
		Alk. Excess of Phosph.				
„ 12th	—	3·15	—	102·.		
„ 13th	350 cc.	3·75	—	101·8.		
„ 16th	—	2·65	—	100	—	No pains.
„ 17th	—	3·1	—	99·2.		
„ 18th	—	3·1.				
„ 19th	—	1·65.				
„ 20th	—	1·65	—	99·8	—	Pudding.
„ 24th	—	3·5	—	—	—	Fish.
„ 26th	—	1·9.				
July 8th	—	1·45.				

The percentage of urea was very high (4·1). The patient was at once placed upon the arrowroot treatment, which he

refused, so that for one day he had no food at all. On the next day, the 24th June, he was placed upon milk diet.

The quantity of the urine oscillated considerably, but was never large. The urea percentage fell gradually day by day, and as it fell the patient got better, the pains left him, and at last when the percentage reached 1·75 he got well.

The total quantity of urea was large, but still not in excess, considering the full diet upon which he had been placed. With this change in his diet and his recovery the quantity fell.

This effect of change of diet upon the urea percentage was as strongly marked as in the other cases, a rise occurring from 4·1 to 4·45, followed by a fall.

Lastly, it is to be observed that the specific gravity varies more definitely with the percentage than with either the volume of urine or the total urea. This is what we should expect to occur.

Case 4.—I have now to refer to the case of John E— (see same table), a fairly severe case of rheumatic fever treated by alkalies and placed upon a diet of milk. Here only the percentage of urea in the *urina sanguinis* was taken. I shall have to refer to this later. At present all I wish to do is to point out how closely the results obtained from this agree with those given by the percentage of the volume during the whole twenty-four hours.

We observe the height of the percentage during the height of the fever, its gradual fall during defervescence, convalescence commencing as soon as the percentage reaches about 1·5, the marked rise produced by the change of diet when fish was given, and the subsequent fall again. Lastly, I have to point out that on July 8th, when convalescence was almost complete—twelve days after the last percentage was taken—the percentage of the urina sanguinis was 1·45, which shows sufficiently clearly the relation the percentage bears to the condition of general health.

From a general review of these four cases I may state that in rheumatic fever, so far as I am able to judge, *the percentage* is very much increased, while I am inclined to

question whether there is any *such* great increase *in the total excretion of urea* as the books state to occur.

Dr. Parkes, in his work on 'The Urine,' quotes Vogel's observations, which give a mean excretion, in three cases of rheumatic fever in men, of 39 grammes, *i. e.* 600 grains, and Brattler's results, quoted also by Dr. Parkes, give an even larger amount, viz. 56·5 grammes, *i. e.* 870 grains, as a mean of four days. In this case the man in health passed 27·4 grammes, or about 450 grains.

I proceed now to the cases of ADDISON'S DISEASE.

CASE 1. A. B— (see Table VII) came into the hospital on July 12th. When admitted she was extremely weak and thin. Her weakness increased rapidly, and after about ten days she died. Her appetite, which had been long failing, was, on her admission, very bad, and for the last few days she was prevented by nausea and sickness from taking food at all.

TABLE VII.—A. B—. Addison's disease.

Date.	Total urine.	P. c. urea.	Total urea.	P. c. ur. sang.	Diet.	
July 12th	700	1·65	11·55	—	Appetite very bad.	N. 70
					Fish,	56
					Wine ʒiv,	—
					Milk Oj.	126
						8½ grms.
„ 13th	900	1·65	13·95.			
„ 14th	700	1·7	11·9	1·725.		
„ 15th	450	1·925	8·66	1·9.		
„ 16th	470	2·	9·4	2·075.		
„ 17th	500	2·125	10·6	2·225	Complete loss of appetite.	
20th	470	*1·775	9·4	2·	*From urine kept twenty-four hours longer than the urina sanguinis.	
Mean.	600	1·83	10·8.			

The mean excretion for the seven days on which observations were made were—

Total vol. of urine.		P. c. urea.		Total urea.
600	...	1·83	...	11·

The total volume is very much reduced, a reduction which was especially marked on the last four days of life.

The percentage, which on the average was about normal, rose towards the last, while the total excretion of urea was greatly diminished, the mean for the seven days being 10·8 grammes. There is to be noticed, with the rise in the percentage at last, a rise also in the total amount of urea, showing that tissue waste was more rapid just before death.

Case 2. In the second case of Addison's disease, that of Emma T— (see Table VIII), we notice similar results.

Table VIII.—Emma T—. Mary Ward. Addison's disease.

Date.	Total urine.	P. c. urea.	Total urea.	Specific gravity.	Diet.	
May 20th .	650 ...	1·9 ...	12·35 ...	— ...	D. L.	N.
					Eggs ij,	150
					Chop,	20
					Brandy ʒiv.	70
						240
						Approx. 16 grms.
„ 26th .	850 ...	1·6 ...	13·6 ...	—		
„ 27th .	780 ...	1·6 ...	12·48.			
„ 29th .	1000 ...	1·65 ...	16·5.			
June 2nd.	600 ...	1·7 ...	10·2.			
„ 4th .	875 ...	1·15 ...	10·06.			
„ 5th .	620 ...	1·4 ...	8·68 ...	1012.		
„ 6th .	550 ...	1·85 ...	10·18.			
Mean . .	740 ...	1·6 ...	12·.			
June 12th .	— ...	1·6 } Urina sanguinis.				
„ 13th .	— ...	1·7 }				

The averages are—

Total vol. of urine.		P. c. urea.		Total urea.
740	...	1·6	...	12·.

The volume of urine is much reduced. The urea percentage is fairly natural, and the total urea much reduced. But both the volume of the urine and the total urea are somewhat larger in this case than in the preceding one, due, doubtless, to its being hardly so late comparatively in in the disease. The urine just before death was not examined. We have only two percentages taken later in life, on June 12th and 13th, and these agree fairly with those previously obtained.

In this case the slight increase in the total amount of urea is probably due to the food (for at this period of the disease there was still some appetite, though it was considerably impaired), especially since it is likely that here the tissue waste was less rapid, the illness in this case having been throughout of a less acute character than in the first case quoted and of longer duration.

The post-mortem examination in this case confirmed the diagnosis.

I have only to add that, in these, as in the other cases, the variations from day to day in the total volume of urine and in the total urea are equally evident, while the percentage remains throughout fairly constant.

The results obtained in these two cases bear out the statements in books, to the effect that the urea in Addison's disease is much diminished, but this diminution I should be inclined to refer almost entirely to the loss of appetite which is so characteristic of the disease. The rate of tissue change, so far as I am able to judge from these cases, and by comparison of these with other cases of partial abstinence from food, is not increased, but remains fully normal until the last, so that the emaciation and loss of strength would be due, not to increased tissue waste, but to diminished tissue repair.

The last case to which I wish to draw attention is one of DIABETES MELLITUS (see Table IX).

TABLE IX.—George F—, æt. 40. Mark Ward. Diabetes mellitus.

(Twelve months ago micturition frequent).

Date.	Total urine.	P. c. urea.	Total urea.	Specific gravity.	Diet.
June 26th	2300 (not all)	1·475	33·925	1037	Appetite very bad indeed. Bran biscuits, Milk Oj, Greens.
„ 27th	2500	1·5	37·5	1036.	
„ 29th	2500	1·475	36·88	—	Full diet.—
July 2nd	3350	1·35	45·2.		Chop,
„ 3rd	3350	1·25	41·88	1032.	Bacon.
„ 4th	3350	1·4	46·9.		
„ 5th	3250	1·6	52·0	—	June 30th.—Eggs iv, 24
„ 6th	3100	1·7	52·7	1032.	hours previously.
„ 7th	3100 (about)	1·45			
„ 9th	2700	1·65	44·55.		
Mean	2640	1·485	39·15.		

He was taken away by his friends in a dying state, and died the next day, July 11th.

The total volume of urine passed in the twenty-four hours is, we notice here, below the average for the disease. Dr. Roberts gives the total volume as varying from 8 to 15 pints (*i. e.* from 4500cc. to 8500cc.), so that here we have a very considerable reduction in the quantity of urine for the day. Variations in this total volume are to be observed similar to those noticed in the other cases.

The percentage of urea is not excessive, but gives an average such as is usual in healthy men upon a moderate diet. It is, moreover, fairly constant throughout, varying from 1·25 to 1·7, the mean being 1·485. I may state here again that the variations in the percentage do not depend upon variations in the total volume of the urine.

We come to that which is the most suggestive feature in this case, viz. the total daily excretion of urea. This is considerably above the average, varying from 33·925 to 52·7 grammes, and giving as the mean 39·15. Variations are to be observed here also in the total amounts from day to day. These variations are due to the compound variations in the percentage urea and the total volume of urine, and there-

fore depend, as do those in the total volume of urine, upon some cause which we do not at present comprehend. The broad fact, then, which we have to observe is the great increase in the total excretion of urea. To what is this increase due?

Urea, being one of the products of the decomposition of nitrogenous substances, has two sources of production—first, the peptones, or products of the digestion of albuminous food, *i. e. the circulating albumen*, and, secondly, the nitrogenous tissue, or what may be called the *fixed albumen*. The increase in the urea in diabetes, which has been a long-recognised fact, has generally been referred to the first of these sources, and connected with the ravenous appetite most diabetic patients have. But in this case it was not so. The diet upon which the patient was placed, as seen in the scale, was rich and highly nitrogenous, yet during the whole time he was under observation his appetite was very bad, and for the last few days of his life food produced so much nausea that he practically took none. We are obliged to abandon, then, this source of the excess of urea, and we are thrown at once upon the tissues as the only other possible source. That there is excessive waste of nitrogenous tissue in diabetes is shown by the extreme emaciation which takes place, and which is due to more than simple loss of fat or drainage of water from the system, by such experiments as these just quoted, and further by the researches of Liebermeister and Reich (quoted in Niemeyer, 'Med.,' vol. ii, p 775), who found that the production of urea in diabetes not only was greater than that of healthy persons moderately supplied with mixed food, but also exceeded that of a healthy man who ate as much food as this diabetic patient did. The amount of urea fluctuated in the patients from 32 to 59 grammes, and in a healthy subject from 29 to 32 grammes. The other explanations of the increased production of urea in this disease we cannot accept, viz.—1st, that it is due to increased metamorphosis of tissue, produced by transudation of water (Roberts on 'Urinary Diseases'), since no such increase is observed in polyuria, where the excretion of urea is, if any-

thing, below rather than above the normal; or 2nd, that it is due to diminished elimination by the skin, for though Liebermeister and Reich show that the proportion in diabetes may be reduced to one third its normal amount, yet, inasmuch as only one thirtieth part of the nitrogen excreted leaves the body by the skin, we have not here shown a sufficient cause to account for an increase which is at least half as much again as the normal. It is right to note, in passing, that this increase has not always been observed, for Dr. Prout ('Stomach and Renal Diseases,' ed. 5, p. 25) states that he met with certain cases in which there was a diminution instead of an increase in the amount of urea. Now, urea is not an absolute measure of the oxidation going on in the body (Voit and Bauer, 'Journ. of Chem. Soc.,' vol. xxiv, Abst.; Voit, 'N. Rep. Pharm.,' xx, 340, 349), for in certain cases of phosphorus poisoning the urea was found to be much increased, while at the same time the amount of carbonic acid evolved was 47 per cent., and that of oxygen absorbed 45 per cent., below the normal.

We may hence have increase of urea without increase of oxidation, which is consistent with other facts in diabetes which seem to point to want of oxidation as a notable feature in the disease, viz. the diminished combustion of the sugar, the commonly low temperature of the patients, and the loss of the power in diabetic patients which healthy men possess of absorbing more oxygen during the night than they need at the time, and of using it up during the day.

We are justified, then, in referring the increased production of urea in diabetes in great measure to decomposition of fixed albumen, of organised albuminous tissue. Can we refer the sugar to the same source?

We will take, to simplify matters, muscular tissue as the type of albuminous tissues. Muscles destroy sugar, as such, in the blood, convert it into glycogen (Bernard, 'Revue Scientifique,' 2me série, tome iv, p. 1022), store it up in themselves, and reconvert it as they require it into sugar. Lactic acid and glycerine are the products of decomposition of sugar (Bernard, *ut supra*, p. 1050), and to these forms

it must be reduced for combustion (Ludwig and Scheremetjewski, Ludwig's 'Arbeiten,' 1868, p. 144). Lactic acid is a measure of sugar decomposition (Bernard, *ut supra*, 1150). Sarcolactic acid is formed by a muscle during contraction (Dubois Reymond, 'J. für Chemie,' 1859, vol. lxxvii, p. 233). Ranke ('Physiologie,' p. 628) has obtained sugar from muscle, after contraction in tetanus, both in the body and out of it.

All these experiments point to the glycogen in the muscle as the source of sugar, or of the acid it produces, so that here muscle is only an apparatus by which sugar is converted and reconverted to meet the demands of the organism.

The fact that glycogen remains in muscle, long after it has disappeared (as the result of starvation) in the liver, shows the extreme difficulty of obtaining proof of production of sugar in muscle independently of the glycogen it contains, and I have been unable to find any exact experiments of the kind to prove it. Yet there are certain experiments which render such a source of the sugar extremely probable.

1st. It is formed in diabetic patients on a pure albuminous diet, *i. e.* from peptones.

2nd. Schultzen and Riess's experiments on phosphorus poisoning lend strong confirmation (Schultzen and Riess, "Ueber Phosphorvergiftung," Separat-abdruck aus der 'Annalen der Charité,' Band x). The albuminous tissues (muscles) undergo extreme fatty degeneration. At the same time urea disappears from urine, but leucin and tyrosin take their place, and a kind of lactic acid (aldehyde of glycerine) appears n the urine (Schultzen, 'Berliner Klinische Wochenschrift,' 1872, No. 35, p. 417).

Here we have just the converse of what we want. In phosphorus poisoning albumen splits up into leucin, tyrosin, &c., and sugar, which is oxidised, and forms lactic acid.

In diabetes, albumen splits up into leucin and tyrosin, &c., which are oxidised to form urea and sugar. In one case the leucin and tyrosin, &c. are oxidised, and in the other the sugar.

This imperfect combustion in diabetes is attributed, not to want of oxygen, but to want of ferment which should prepare the sugar for oxidation.

3rd. Lastly, we have the experiments of Dr. Sydney Ringer ('Trans. Med.-Chir. Soc.,' 1860, p. 323), who found in starving animals a definite relation between the amount of urea and sugar, the proportion being 1 to 2·35, and that upon a purely flesh diet this proportion was maintained, showing that in both cases the albumen must have split up into urea on the one hand and sugar on the other.

It is not in all cases of diabetes that emaciation or loss of tissue is a marked feature. Are there any facts which suggest a possible cause for such rapid tissue waste, or hint at any possible way in which it could be produced?

i. Peptones, we know, give rise to sugar and urea (Dr. Sydney Ringer, (*ut supra*).

ii. Hypertrophy of the pancreas is observed in certain cases of diabetes (Niemeyer, 'Med.,' *ut supra*).

iii. Pancreatin is an active ferment in the digestion of albumen (Hermann, 'Phys.,' p. 111).

iv. It is active (unlike pepsine) in an alkaline fluid (Hermann, 'Phys.,' p. 111).

v. Pancreatin has been found in every tissue in the body.

Hypertrophy or over-activity of the pancreas would cause increased production of pancreatin. This would be carried all over the body. Being in an alkaline fluid—the blood—it would be in a condition to act upon the albuminous tissues. These it would convert from the insoluble to the soluble form, producing peptones, which the diabetic diathesis, to whatever cause that may be due, would split up into urea and sugar. So that we might assert the emaciation and loss of tissue in diabetes to be due to a process of *self-digestion*, the products passing out of the body by the urine. This, of course, is pure hypothesis, and must be

[1] Hermann ('Physiol.,' p. 43) "Es ist der Zusammensetzung nach sehr leicht möglich dass Fette, Glycogen, Zuckerarten aus der Eiweisskörpern hervorgehen."

taken simply for what it is worth; but it serves to bind together a certain number of facts hitherto independent in the pathology of diabetes.

Professor Haughton's authority ('Phenomena of Diabetes Mellitus,' Dublin, 1871) lends support to such a view of the splitting up of albumen.

Dr. Brunton, in his paper ('Brit. Med. Journal,' 1873 and 1874), tabulates the causes of diabetes in the following way:—Diabetes may arise, i. from increased formation, and ii. from diminished combustion of sugar. To take the last first—

From diminished combustion, due—

(*a*) To insufficiency of the ferment which should convert sugar into lactic acid and glycerin.

(*b*) To an altered quality of the sugar, which enables it to resist the action of the ferment.

(*c*) To diminished circulation through the muscle preventing the sugar from coming sufficiently into contact with the ferment.

From increased formation, due—

(*a*) To excessively rapid digestion of starch or sugar.

(*b*) To failure of glycogenetic function of the liver, and possibly of the muscles also.

(*c*) To increased transformation of glycogen into sugar, due (1) to accelerated circulation through the liver; (2) to a larger proportion of ferment (*a*) in the organ, (*b*) in the blood.

To these I would beg permission to add a fourth—

(*d*) To the direct decomposition of albuminous tissue into urea on the one hand and sugar on the other.

I have now placed the Society in possession of all the cases I propose to bring before it this evening, and it remains only to consider what conclusions these cases suggest. I will first speak of the results in their general aspect, then in their relation to states of fever and convalescence, and lastly I will endeavour to indicate the direction in which the observations made seem to point.

And in the first place with reference to the relation of

the total volume of urine, the percentage of urea, and of the total urea to each other.

Water has four main channels provided for its elimination from the body—the kidneys, the skin, the lungs, and the intestines, and hence the total volume of urine will vary inversely with the compounded variations of the other three, and the activity of each and every one of these organs of elimination is subject to very considerable vicissitudes in dependence upon varying influences both internal and external. So that we should expect, *a priori*, very considerable variation to occur in the total volume of urine in different individuals, and in the same individual at different times, and this we find to be the case in the observations made, and, until we are able, which at present we are not, to define the exact part each of these organs takes in the excretion of water, we cannot fairly expect to derive any but the most general information of clinical value from consideration of the variations in the total volume of urine. (In Bright's disease, however, these variations assume a clinical importance which does not belong to them in other diseases.) To this conclusion I have drawn attention frequently in speaking of the separate cases, and I may refer for confirmation to a case of aneurism (see Table X), treated on Mr. Tufnell's plan, in which the diet was known and could be tolerably accurately estimated. This case I shall discuss in another place, and I do not propose to-night to do more than use it for reference, according as I find it convenient for illustration.

TABLE X.—Thomas S—. Aneurism.

Date.	Total urine.	
July 9th	450	... ʒviij of liquid.
„ 10th	450.	
„ 11th	475.	
„ 12th	350.	
„ 13th	600.	ʒx of liquid, varying from
	400	... 520 cc. to 600 cc.
	250.	
	550.	

Date.	Total urine.
July 17th	525.
„ 20th	400.
„ 21st	500.
„ 22nd	450.
„ 23rd	475.
„ 24th	550.
„ 25th	500.
„ 26th	400.
„ 27th	300.
„ 28th	300.
„ 29th	425.
„ 30th	400 (?).
„ 31st	575.
Aug. 1st	475.
„ 4th	550.

The quantity of water ingested was constant, being eight ounces above the line and ten ounces below, and in spite of this the variations to which I have referred are very considerable. I may state in passing that these results may be accepted as trustworthy, for I took every pains to ascertain each day that the total quantity was passed.

The *total volume of urine* will, then, we are forced to conclude, give us very little information of value. From the *percentage of urea*, however, I think we can obtain more definite results. Given fairly constant conditions of disease or health, the percentage urea remains fairly constant. Any sudden alteration in these conditions causes alterations in the percentage.

In all the cases of fever I have quoted, during the fever the percentage remains high and constant. In Ellen C—'s (Table I) case the average is 3·5, with which, as I have stated, nearly all the experiments agree. So also in E—'s case (Table II), in John E—'s (Table VI), and in G—'s (Table III). With the termination of the pyrexia the percentage falls, but of this I shall speak more in detail when I reach the question of fever. Small variations of temperature, however, do not, in the cases I have quoted, seem to me to produce similar changes in the percentage. Nor does the percentage vary inversely with the volume of

urine, and this agrees with the result of physiological experiments which show that with increased volume of urine there is increased excretion of urea (Kaupp, 'Archiv. f. Phys. Heilkunde,' 1856). Nor, indeed, is the percentage dependent in any way upon the total amount of urea excreted, a conclusion which, indeed, follows from the statement above.

As instances of the effect produced upon the percentage by sudden alterations in the conditions of life, I may quote the sudden drop observed at the termination of pyrexia, and, again, the effect of alterations of diet, which produce at first a sudden rise followed immediately by one or two oscillations in the percentage, which then becomes again constant. See Ellen C—'s case on June 1st and on June 13th; James C—'s case on June 7th and June 8th; William T—'s on July 8th and 9th; John E—'s on June 24th; and Thomas S—'s case on July 9th.

Lastly, I may state that in health the percentage appears to vary fairly definitely with the specific gravity of the urine, a fact of some importance in pathological conditions to which I shall have again to refer.

I will defer what more I have to say as to the information to be derived from the consideration of the urea percentage until I discuss it in its relations to fever and convalescence.

There is one great practical difficulty which attends all quantitative examinations of the urine of the whole day, and this is its collection. It is necessary to depend so much upon the nurses and the patients that it is only in very carefully conducted observations, and at cost of considerable watchfulness on the part of the observer, that trustworthy and accurate results can be obtained. This objection applies no less to the percentage of the total urine than to the volume. If, then, it could be shown that whatever practical information can be derived from the percentage of the total urine can be equally well obtained from the percentage of a part of the urine, an immense difficulty would be overcome in investigations much facilitated. The urina sanguinis, to show, gives a percentage which bears so

close a relation to the percentage of the total urine of the day as to justify us in accepting this as a sufficient substitute for the latter.

In the second case of Addison's disease quoted the percentage of the total urine of eight days during the early part of the illness gave a mean of 1·6 grammes. On June 12th and 13th the percentage of the urina sanguinis was taken. This gave 1·6 and 1·7, the mean being 1·65, which agrees very closely with the average percentage of the previous experiments.

Again, in the first case of Addison's disease, we have these two series of percentages (see Table VII):

	P. c. of total urine.	P. c. of urina sanguinis.
July 14th	1·7 ...	1·725.
„ 15th	1·925 ...	1·9.
„ 16th	2· ...	2·075.
„ 17th	2·125 ...	2·225.
„ 18th	(?) 1·775 ...	2.

Also in the cited aneurism case we have the following series of percentages (see Table XI):

Date.	P. c. of total urine.	P. c. of urina sanguinis.
July 9th .	4·7 ...	4·7.
„ 10th .	4·85 ...	4·9.
„ 11th .	4·95 ...	5·1.
„ 12th .	5·25 ...	5·4.
„ 13th .	5· ...	5·25.
„ 14th .	5·2 ...	5·18.
„ 15th .	5·55 ...	5·385.
„ 16th .	5·25 ...	5·25.
„ 17th .	5·3 ...	5·4.
„ 20th .	5·025 ...	5·1.
„ 21st .	4·5 ...	4·425.
„ 22nd .	4·53 ...	4·575.
„ 23rd .	4·65 ...	4·65.
„ 24th .	4·35 ...	—
„ 25th .	4· ...	—
„ 26th .	4·875 ...	5·175.
„ 27th .	4·4 ...	4·6.
„ 28th .	4·8 ...	4·4.

Date.	P. c. of total urine.	P. c. of urina sanguinis.
July 29th	4·6	4·9.
„ 30th	4·56	—
„ 31st	4·1	4·425.
Aug. 4th	4·05	4·2.

In some instances in the last series of observations the figures in the two columns agree, while in none is this difference very marked, and throughout it is to be borne in mind that, from the extreme concentration of the urine, the height of the percentage, and the dilution required for the convenience of manipulation, the errors of experiments are here multiplied threefold. At any rate, a number of consecutive experiments which agree so closely as these do appear to prove conclusively the point upon which I am insisting. I would refer, lastly, to John E—'s case (Table VI), in which the percentages of urina sanguinis alone were taken. It will be observed, on comparing these with the percentages taken in the other cases of rheumatic fever, that we have here a similar series of percentages with similar variations produced by similar change in the condition or diet of the patient. We have the high percentage of fever, the sudden fall of convalescence, and the oscillation due to change of food.

I have some hesitation, however, at present, in stating that such a definite relation exists between the percentage of the urina sanguinis and of the total urine of the day in persons in robust health, and not just recovering from illness, for, though I think it extremely probable that even here a similar relation exists, I have not yet sufficient observations to decide the question.

The facts stated above justify the conclusion, I think, that whatever practical information can be derived from the percentage of the total urine of the twenty-four hours can equally well be derived from the percentage given by the urina sanguinis.

As regards *the total urea* excreted, if what I have said with reference to the constancy of the percentage and the indefinite variations in the total volume of urine be correct, it is evident that we cannot expect to derive very much

information from day to day from the total amount of urea, this being the product of two factors, one of which has been shown to vary indefinitely and according to no law which we are at present able to determine. As to the kind of information which we may hope to derive from the total amount of urea, I shall speak again later.

So far, then, it seems probable that it is to the percentage, and not either to the total volume of the urine or to the total excretion of urea, that we must look for the chief indications of clinical importance.

I wish now to draw attention to the results which these observations suggest as to the pathology of urea in the two conditions of fever and convalescence. I may merely state, in passing, with reference to the *total volume of the urine,* that, beyond the broad fact that in fever the volume is much reduced, and that it gradually rises to the normal as convalescence advances, we appear to be at present able to draw no other conclusion of clinical importance. The *percentage,* however, gives us a very interesting series of results, but, as I have referred to these in discussing the particular cases, I need only here briefly recapitulate what has been observed.

i. During fever the percentage is high and remains fairly constant as long as the fever lasts.

ii. During the fever it does not follow the slight variations of temperature.

iii. Nor does it depend on the volume of the urine.

iv. Changes in diet cause, during the fever, the same oscillations as they cause during convalescence.

v. With the termination of the fever the percentage falls.

vi. Convalescence may be divided into two periods.

vii. The first period is characterised by a gradual and progressive fall in the percentage, even until it is considerably below the normal.

viii. The second period is characterised by a gradual and progressive rise in the percentage, until at last a mean varying from about 1·5 to 2·0 is established, when it becomes constant.

ix. There is between this specific gravity and the percentage of urea during convalescence generally a fairly definite relation.

It remains now to consider what conclusions can be drawn from the *total amount of urea*, and as I have already dwelt upon them, I need here again only briefly sum them up.

During the fever—

i. The total amount is much reduced, and is not in large excess (as stated commonly in books).

ii. The total amount varies very considerably from day to day, independently of any law which we can determine at present.

iii. These variations are independent of the diet.

iv. And also of the temperature, hence the total urea is not a measure of fever.

v. The variations depend upon the total volume of the urine rather than upon the percentage urea.

vi. The variation was considerable in the different cases quoted.

During convalescence—

vii. The total amount is often extremely reduced.

viii. It gives an average which is different in the two periods of convalescence, but sometimes that of the first period is higher, and sometimes lower, than that of the second period. This may be accounted for, perhaps, by the different fevers from which convalescence is taking place.

	First period.		Second period.
— Pneumonia . . .	17·7	...	23·0.
— Rheumatism . . .	21·74	...	—

ix. The same variations, independent in great measure of the diet and of the percentage, and varying with the total volume of the urine, are to be noticed during convalescence or during fever.

It is commonly stated that urea is the measure of tissue change, and also that its excretion is governed by the same laws in health and in disease (Neubauer and Vogel, 'Analyse

des Harns,' p. 338). With these statements the results of the present observations seem hardly to agree, for it has been pointed out that the relation between the amount of nitrogen in the food and that estimated in the urine as urea is by no means so close as it is asserted to be in health. This conclusion is more definitely and clearly shown in the case of aneurism to which I referred before.

Again, as regards tissue change, which must necessarily be in health constant, if it be true that the excretion of urea varies directly with the amount of nitrogen in the food, it is not possible to trace here any proportion between the amount of urea excreted and the amount of tissue change which we know from other sources must be going on. But, apart from the tissue change which we know to be so much increased in fevers, we have not in these cases sufficient urea to account for the nitrogen of the food alone, a fact which of itself is sufficient to show how changed the relation of urea to its source, the tissues and the food, must be in disease. And I may refer, in confirmation, to the two cases quoted of fatal pneumonia, in which there is certainly no great excess, if any, in the amount of urea excreted, although we have in these two cases extreme nervous excitement and high fever, conditions each of which is said by itself to produce increased elimination of urea. Further, it is stated that, under similar conditions in the same person, the quantity of urine passed in the day remains fairly constant. This, again, the observations now made do not appear to support. In convalescence, however, the present results agree fairly with the statements made by Neubauer and Vogel ('Analyse des Harns,' p. 338).

In speaking of the cases of pneumonia I endeavoured to point out that the peculiar diminution in the amount of urea excreted was probably due to some interference with the process of oxidation, and I quoted Schultzen and Riess's experiments to show that albumen is normally split up into leucin and tyrosin, &c., and that these are subsequently oxidised to urea; but that, if the oxidation process was interfered with, urea disappeared from the urine, and leucin

and tyrosin, &c., took its place. The diminution of urea, therefore, from the normal amount excreted in health may perhaps give us a fair means of estimating the amount of interference which exists with the normal nutrition and oxidation of the tissues. The other explanation offered of the facts was that these products, instead of being excreted, were stored up, as such, in the body, and were subsequently, during convalescence, excreted. This explanation was supported by a case quoted by Dr. Parkes, in which he observed a critical discharge, during convalescence, of uric acid. We are justified, I think, in the absence of direct experiment, in assuming that it is by the urine that any excess of nitrogen during fever leaves the body, but that if so it must be in some other form than urea, probably leucin and tyrosin, as suggested above.

This conclusion, in the particular case of pneumonia quoted, is supported by the want of relation between the specific gravity of the urine and the amount of urea contained in it, a difference which suggests the presence of a large amount of solid apart from the urea, and which is applicable on the above assumption. The specific gravity is, of course, but the roughest guide. That which is really required is a comparison between the amount of urea and the total nitrogen of the same urine. At present we have, I believe, no definite experiments of this kind, and, important as the results obtained from the estimation of the amount of urea in urine are, yet far more accurate and important results would, I believe, be obtained from an investigation into the variations of the total nitrogen. Until we have such experiments, and are able to compare the total urea with the total nitrogen in any urine, all interpretations of the total urea excreted will, I think, be unsafe and unsatisfactory.

A CASE

OF

LEFT SUBCLAVIAN ANEURISM

EATED BY TEMPORARY COMPRESSION APPLIED DIRECTLY TO THE ARTERY IN THE FIRST PART OF ITS COURSE; WITH REMARKS.

BY

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COMMUNICATED BY
JOHN WOOD, F.R.S.

(Received February 5th—Read April 27th, 1875.)

L—, æt. 35, unmarried, a laundress, first came under
in August, 1872. She was at that time a patient
General Infirmary. She was suffering from a
urism on the left side, which she had noticed
y, and which she attributed to a blow received
he previous Christmas (1871). The treat-
digital compression applied to the third
n artery for twenty-four hours consecu-
ent followed this treatment. In April,
under ny care. The aneurism had
d much from pain in the
o treat her by galvano-
mended by Dr. John

Duncan, of introducing both the negative and positive needles into the sac. As the details of this treatment have been already published,[1] it is unnecessary for me to repeat them here. It is sufficient to remark that the operation was repeated five times with great temporary benefit, but without permanent cure. On the last occasion, February 24th, 1874, I combined the treatment with manipulation, but without any apparent result.

In November last she again applied to me. She complained much of a return of pain in the shoulder and arm. On examination it was seen that the position of the aneurism had considerably altered, that it had extended upwards above the clavicle into the neck, and that in that position it was approaching the skin. Below the clavicle, at the seat of the former operations, the pulsation was slight, and the tumour felt hard. Her general health was beginning to suffer. she was losing flesh, her appetite was bad, and she never slept without the aid of an opiate. Wishing to watch the further progress of the case, I advised delay, but at the end of a month, as her condition was getting worse, I requested my colleagues, Messrs. Wright and Horsfall, to see the patient with me. At that time pulsation could be felt extending upwards from the centre of the clavicle for an inch and a quarter, and downwards and outwards from the same spot for two inches and a half; the border of the tumour could be plainly felt internally above the clavicle, extending inwards under the outer part of the sterno-mastoid muscle, while outwards there was not a distinct spot at which the swelling stopped, but it seemed to extend towards the axilla, and to be lost in the upper part of that space. I proposed the operation of temporary compression of the artery in the first part of its course (*i. e.* internal to the scaleni muscles) and my colleagues, after careful examination and discussion, although by no means sanguine as to the result, agreed with me that the operation was justifiable, and promised me their assistance should I determine to undertake it. For the

[1] 'The Lancet,' July, 1874.

reasons stated below I made up my mind to give the patient this last chance of life, and on January 2nd of the present year (1875) performed the operation.

The patient being placed under chloroform, and having her shoulders supported by pillows and her head thrown back, an incision was made along the anterior border of the sterno-mastoid from the centre of the sternum upwards for two inches; this was joined by another of the same length along the clavicle. The flap thus formed, consisting of skin, superficial fascia, and platysma, was reflected; a small vein above the clavicle was ligatured, and, a director being passed under the sternal origin of the sterno-mastoid, this part of the muscle was divided. In doing this a large vein, the anterior jugular, was opened, and was secured by a ligature. The inner half or more of the clavicular origin of the muscle was then divided. The sterno-hyoid and sterno-thyroid muscles were thus exposed, and the internal jugular could be seen on the outer side of these muscles. The knife was now abandoned. Helping myself with a director, I slowly passed my finger through the fat and cellular tissue situated on the outer side of the vein, and reached the inner border of the anterior scalenus. Introducing a retractor into the wound, I hooked the jugular towards the middle line, and saw a large vein and what was apparently a small empty vein (thoracic duct?) passing over the aperture made by my finger; these were then placed under the retractor. The finger was now passed downwards, following the scalenus muscle; the phrenic nerve was reached and then the first rib. I now expected to find the artery directly behind the muscle, but on feeling was unable to detect any pulsation. The artery was obviously displaced from its normal position, and it was necessary to search for it. This was a somewhat tedious process. The carotid artery, the subclavian vein, the internal mammary artery, the transverse process of the seventh cervical vertebra, the aneurism itself, and the pleura were all reached in turn. At last, when my finger was inserted for its whole length, I felt obscure pulsation, apparently through a layer of pleura; by the help

of a director the artery was exposed and an aneurism needle (Gibson's) was passed round it. Pressure on the artery now stopped all pulsation in the aneurism. In passing the needle a small opening was made in the pleura. I now attempted to apply to the artery a pair of forceps which I had had made for the purpose; they, however, were not long enough, but a pair of ordinary torsion forceps were applied with ease. The wound was brought together by a few points of suture, and the operation completed at 2 p.m. One third of a grain of morphia was given hypodermically.

At 5 p.m. the pulse was 130, small, and irregular; respirations 44, the air passing in and out of the pleura at each breath. The left side of the chest was hyper-resonant, the breathing distant; on the right side loud mucous râles were heard. She rallied somewhat by 8.30, the pulse having a better character.

At 12.20, midnight, owing to her feeble condition, it was deemed expedient to remove the forceps and close the wound, thus preventing the ingress of air into the pleura. This was easily accomplished; the aneurism felt quite hard and contracted; no pulsation could be detected in it or in the left radial artery.

January 3rd, 9 a.m.—The general condition is improved. Pulse 116; temperature 102·8°; respirations 36. The aneurism felt hard; slight pulsation is detected in it above the clavicle. She passed a restless day, being troubled by a continuous hacking cough. In the evening the pulsation could still be felt in the aneurism, and also feebly at the wrist.

4th.—Has passed a quiet night; the pulsation has disappeared, both in the aneurism and at the wrist. From this time till the morning of the 7th she gradually improved. Her pulse varied from 112 to 130; temperature from 99·6° to 101°; respirations from 25 to 40. There was no return of pulsation in the aneurism or at the wrist.

On January 7th severe pain in the side supervened; her cough became more troublesome, and she expectorated large quantities of bronchitic sputa. She died at two

o'clock on the morning of the 8th, having lived five days and a half.

The friends strongly objected to a post-mortem examination. After much difficulty I obtained leave to examine the wound. The incision was prolonged along the clavicle, and this bone, being disarticulated at the sternal end, was thrown outwards; the cartilage of the first rib was then divided. The wound had united in the greater part of its extent. On opening the pleura behind the first rib about a pint of serum escaped from its cavity. Passing the finger upwards and backwards, a small opening, which would not admit the tip of the finger, was detected at the apex of the membrane. The artery had been compressed just before the origin of the vertebral; it was patent and in a perfectly healthy condition. The aneurism was long and fusiform, commencing a quarter of an inch from the thyroid axis, and extending outwards for three inches and a half. The artery on the cardiac side was of normal size, but at the peripheral extremity was somewhat dilated. The whole was filled with a hard firm clot, and it was, in fact, cured.

Remarks.—Before determining to perform the operation described above, I felt it was necessary to determine three points:—(1) Was there no other method of treatment that could be adopted? (2) Was the proposed operation practicable? and (3) Was there a reasonable prospect of success?

(1) There are numerous methods by which an aneurism of this description may be treated. The chief are pressure, manipulation, galvano-puncture, distal ligature, distal ligature combined with amputation, medical treatment, and ligature on the cardiac side. Of these, pressure, manipulation, and galvano-puncture had been tried, the two former without result, the latter with great benefit, which, unfortunately, was only of a temporary character, and which did not result in a permanent cure. Distal ligature without amputation would give small prospect of success; the patient would not hear of amputation, though at one time I thought seriously of the operation, encouraged by the case

of partial success recorded by Professor Spence. Medical treatment could not be tried, as the patient's position would not allow her to take the requisite time which must of necessity be spent over the method of treatment. Nothing then was left but to apply a ligature or some substitute for it to the artery on the cardiac side of the aneurism, that is to say, internal to the anterior scalenus in the first part of its course.

(2) The opinion expressed by the majority of surgeons is that this operation is "impracticable." For instance, M. Erichsen says,[1] "On the left side this operation (*i. e.* ligature) is not practicable on account of the depth at which the artery is situated;" again, Mr. Colles,[2] who was the first surgeon who tied the subclavian artery in the first part of its course, says, "This operation, difficult on the right side, must be deemed impracticable on the left." Many other authors could be quoted who express the same opinion. As, however, Dr. Rodgers, of New York, performed this operation in 1846, it was plain that these opinions were fallacious. After making many dissections of the part on the dead subject, both injected and uninjected, I came to the conclusion that the operation on the dead body was attended by no great difficulties. The thyroid axis was easily found in the interval between the anterior scalenus and internal jugular vein, and this served as an excellent guide to the artery, which could in a few minutes be exposed without injury to any important structure.

(3) The operation being practicable, was there then a fair prospect of success? If the ligature were applied in the usual manner the answer would be distinctly No. The one case in which this operation was performed died of secondary hæmorrhage on the fifteenth day. The ten patients on whom the right subclavian had been tied in the first part of its course all died. These results were not encouraging. The cause of death in ten out of the eleven cases is recorded

[1] 'Erichsen's Surgery,' vol. ii. p. 83.

[2] Quoted in 'Holmes' System of Surgery,' vol. iii, p. 618.

as due to hæmorrhage,[1] but no less than nine of the cases lived for ten days and upwards, one reaching the beginning of the sixth week. It was not unreasonable, then, to conclude that if secondary hæmorrhage could be avoided the operation, instead of being universally fatal, would present a fair proportion of successful results. The ligature being, then, inapplicable, I determined to try the effect of direct pressure on the artery, intending to keep up this pressure for twelve hours, or possibly for a longer time. The experience of Dr. Murray, of Newcastle,[2] seemed to show that the treatment held out every prospect of success, and a case which I had the advantage of seeing in the Leeds Infirmary, under the care of Mr. Wheelhouse, confirmed me in this opinion. This case was reported to the Clinical Society during their last session.[3] The patient was cured of an inguinal aneurism by six and a half hours' pressure on the abdominal aorta under chloroform. The patient died some months after of another disease, and it was found that the aneurism was quite solid, and was fast being removed by absorption, while the aorta at the point of pressure was in its normal condition. It was shown by this case that to cure an aneurism it was unnecessary to occlude the artery. The complete temporary arrest of the circulation through the aneurism was sufficient to effect a cure, and this might, I thought, be effected without making the pressure so tight as to endanger sloughing at the point of application.

The *modus operandi* of this method of treatment deserves a careful consideration. It is evident that the cure is not effected by the gradual deposition of fibrin on the walls of the sac, the slow deposit being due to the passage of a small quantity of blood through the tumour. In a case of this sort the coagulation is sudden, and probably commences soon after the application of the pressure. The aneurism is filled by a clot formed out of the blood that was in it at the

[1] Erichsen, op. cit., p. 83.

[2] 'Medico-Chirurgical Transactions,' vol. xlvii, p. 187.

[3] 'Clinical Society's Transactions,' vol. iii.

time the pressure was applied. This clot, like any other in or out of the body, slowly contracts; for a time the walls of the sac contract round it, but in some cases it will shrink more quickly than the walls, and the pressure being removed from the supplying artery, the blood will pass between the clot and the wall, causing a return of pulsation similar to that which occurred in the case under consideration.[1] The blood will, however, have much difficulty in passing through the limited space left in the sac; fibrin will be deposited from it and the cure completed.

The death of my patient is to me a source of much regret. It was undoubtedly caused by the wound in the pleura, and this, owing to the displaced position of the artery, was, I believe, unavoidable.[2] If this wound had not been made, there seems every probability that the patient would have survived the operation, and if so the aneurism would have been certainly cured. Notwithstanding the unsuccessful result, I still believe this operation to be right in principle and likely to be successful in practice. By applying temporary compression in the place of the ligature it is at any rate possible that these operations for subclavian aneurism may become more successful in the future than they have been in the past. For my own part, I shall seize any opportunity that may occur of again putting this method of treatment into practice, and that not only on the subclavian arteries, but on any of the larger arteries of the body. I can only trust that on the next occasion I may meet with a more successful result.

[1] In Mr. Wheelhouse's case a similar pulsation was observed.

[2] Dr. Rodgers also made a wound into the pleura; it was not apparently followed by evil consequences.

ON THE

HISTOLOGY OF THE SO-CALLED NUTMEG LIVER.

BY

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(Received February 5th—Read May 11th, 1875.)

If the liver of a healthy animal be examined immediately after death it will be found to present a uniform red-brown appearance. Soon after, however, the uniform appearance is lost. The surface becomes mottled, and the mottling on further examination will be found to correspond closely with the distribution of the acini of the liver. The circumference of the acinus is pale, while the centre is deeper coloured. The cause of this unequal distribution of colour is to be found in the arrangements of the blood-vessels; the portal vein, which within the liver closely resembles an artery, is distributed to the circumference of the lobule, and, like an artery, empties itself of blood soon after death. The hepatic vein, on the other hand, which arises in the centre of the lobule, remains full of blood, as is the rule with the ordinary systemic veins.

It is an exaggeration of this mottling to which the name of nutmeg liver has been given. Whenever there exists a [illegible]ndrance to the return of blood from the hepatic vein, [illegible]state of the liver may very readily be set up. The [illegible] of the hepatic vein become gorged with blood, and

a deep brown-red colour is thus given to the centre of the lobule; while the outer part of the lobule, to which the portal vein and hepatic artery are supplied, remains pale. Hence a deep contrast between the centre and circumference of the same lobule.

Kiernan described nutmeg liver under the name of the second stage of congestion, or of active congestion of the liver. "It very commonly attends," he says, "disease of the heart and lungs; the congestion begins in the hepatic veins, and extends towards the portal veins."[1] He has thus accurately enough described the pathology of the disease, so far as it relates to the blood-vessels; before him it was well-nigh impossible to be explained.

Cirrhosis of the liver and nutmeg liver have not always been distinguished. It is clear that five-and-thirty years ago the two morbid states were confounded. Becquerel, in analysing his cases of cirrhosis, sets down heart disease as a cause in exactly one half.[2] So more recent writers, as Rokitansky[3] and August Foerster,[4] have mentioned heart and lung disease amongst the causes of true cirrhosis.

Budd appears to have been one of the first to separate cirrhosis and nutmeg liver, and on the ground that simple passive congestion of the organ could never lead to an active inflammation.[5] Dr. Handfield Jones a few years later expressed his opinion that nutmeg liver does not terminate in the contracted hobnail cirrhosis. As this writer's description of the state of the connective tissue in nutmeg liver seems to me to agree more closely with the facts of the case than those of many other writers, it may be worth while to detail his views more at length. Dr. Handfield Jones regards, as the essential circumstance of the nutmeg liver, the effusion of an unhealthy plasma, not only in the canals and fissures of the liver, that is, in the capsule of Glisson,

[1] Kiernan, 'Phil. Trans. of the Royal Society of London,' 1833, p. 754.
[2] Becquerel, 'Arch. gén. de Méd.,' 1840, t. viii, p. 40.
[3] Rokitansky, 'Handb. der path. Anat.,' Wien, 1842, Bd. iii, p. 347.
[4] August Foerster, 'Handb. d. spec. path. Anat.,' Leipzig, 1854, p. 109.
[5] Budd, 'On Diseases of the Liver,' London, 1845, p. 118.

but in the external part of the lobules. The investing membrane of the lobules is very greatly increased, and becomes much more condensed and more distinctly fibrous. The effused plasma likewise seems to insinuate itself between the cells in the external part of the lobule.[1] With the description of the changes in the lobule itself I am less able to agree. Liebermeister was, I believe, the first to acknowledge that Dr. Jones' view of the changes in the capsule of Glisson was correct, and he has devoted a long chapter in his book on diseases of the liver to this subject.[2] But, so far as I know, few writers have followed in his steps, Dr. Henry Green being one of the few who describes an interlobular growth as being constantly seen in nutmeg liver.[3] Rindfleisch[4] and Klebs[5] certainly speak of an interlobular growth, as being sometimes seen, but they evidently look upon it as an accident not to be associated with the essential phænomena of nutmeg liver.

The prevailing doctrines as to the histology of nutmeg liver are, however, altogether different from that of Dr. Handfield Jones. They are those expressed by such authorities as Virchow,[6] Frerichs,[7] and Rokitansky,[8] and may be stated as follows: that the cells of the liver in the centre of the lobules atrophy from the pressure of the dilated vessels; that their place in the centre of the lobules is then taken by a highly vascular tissue, formed of capillaries and new connective tissue, while the cells of the circumference of the lobules and the inter-lobular connective tissue remain unaltered. My observations have led me, however, to take a very different view of the

[1] Handfield Jones, 'Medical Gazette,' 1848, vol. vii, p. 1033. It is an abstract of a paper read before the Pathological Society of London.

[2] Liebermeister, 'Beiträge zur path. Anat. und Klinik der Leberkrankheiten,' Tübingen, 1864, p. 77.

[3] T. Henry Green, 'An Introduction to Pathology and Morbid Anatomy,' London, 1871, p. 244.

[4] Rindfleisch, 'Lehrbuch d. path. Gewebelehre,' Leipzig, 1867, p. 383.

[5] Klebs, 'Handb. d. path. Anat.,' Berlin, 1869, p. 424.

[6] Virchow, 'Würzburger Verhandlungen,' 1854, Bd. iv, p. 349.

[7] Frerichs, 'Klinik der Leberkrankheiten,' Braunschweig, 1858, Bd. i, p. 374.

[8] Rokitansky, 'Lehrbuch der path. Anat.,' Wien, 1861, Bd. iii, p. 247.

disease, and have caused me to support in general the statements of Dr. Handfield Jones and Professor Liebermeister.

The observations which follow were made upon twenty livers which showed a well-marked nutmeg appearance, met with in the post-mortem room of St. Bartholomew's Hospital during the last two years. Parts of the livers were either hardened altogether in weak chromic acid, or first kept for a few days in weak chromic acid and then put into spirit of wine. The thin sections were in all cases coloured with carmine and mounted in glycerine. The following descriptions must be understood of sections examined with Hartnack's microscope, ocular 3, objective 9, *à immersion :*

In the earlier stage of the disease the chief morbid appearance is to be found in the centre of the acinus, and is caused by a dilatation of the radicles of the hepatic vein. In the section these vessels may be seen distinctly enlarged and still completely filled with red corpuscles. Their appearance varies according to the direction of the section of the blood-vessels. When the cut has fallen lengthwise upon the vessels, they are seen as long channels passing between the liver-cells; when the vessels are cut directly across, they are then surrounded by a ring of liver-cells varying in number. The liver-cells themselves seem to be at first not much changed in character. Their outline is, however, hard to make out, so that they cannot be readily counted; they show a large nucleus and granular contents.

In the later stages the dilatation of the capillaries becomes very great. The vessels in the centre of the acini are dilated to twice or three times their natural size, and in this way grave changes are wrought upon the tissues immediately around them. The rows of liver-cells between the vessels, at first only somewhat lengthened and flattened, become so much pressed upon that their shape becomes almost linear; a nucleus can scarcely be made out, and their contents are seen to be dark and granular.

The latest stage of all is that in which the centre of the lobule becomes little more than a network of vascular tissue,

between the meshes of which a few highly granular and pigmented liver-cells lie. On looking at specimens in this stage with a low power (Hartnack, oc. 3, obj. 4), the outer part of the acinus is seen to be formed of a ring of almost colourless liver-cells, while the centre is filled with a red tissue, studded with pigmented liver-cells; and this tissue, as seen with a low power, looks very like connective tissue. But on examining the specimen with higher powers, this tissue is found to be made up of channels, the walls of which are exceedingly delicate and transparent, and within which red blood-corpuscles can yet be made out. No nuclei can be seen in the midst of this tissue as in the interlobular tissue, but studding the field are numerous rounded or slightly polygonal cells, with highly pigmented contents, and containing no discoverable nucleus. These cells I hold to be the remains of liver-cells.

This last stage of the disease is rarely met with, but it is clear that it is this stage which Rokitansky has endeavoured to portray in the woodcut which shows an intra-lobular vessel surrounded by a highly nucleated connective tissue, in which a few pigmented liver-cells are seen. I am quite unable, however, to agree with the statement that the tissue in the centre of the lobule is an overgrowth of the connective tissue. It is rather to be regarded as the transparent wall of the vessels. The only other change which I have been able to make out is a thickening of the wall of the hepatic vein in the centre of the lobule.

These changes in the vessels and cells rarely go beyond the middle zone of the acinus; I have not found them exceed this in any of the specimens which I have examined. Fatty infiltration of the cells of the outer border of the lobule is not so common or so usual as from the writings of others I had been led to believe. In a very few only did the amount of fat in these cells exceed what is natural. In the few cases in which there was more fat than is common the excess was very great, and the border of the acinus showed cells filled with large fat drops for some depth towards the centre.

The connective tissue of the capsule of Glisson is the seat of important changes. The centre of the acinus is not the only part of the liver which suffers; a large part of the connective tissue of the liver becomes actively engaged in the disease. In the earlier stage the capsule of Glisson may be found increased in size and with numerous lymphoid corpuscles present in it. This may be seen very early. In a child of eight years, in whom, so far as could be made out, the heart disease has lasted only three weeks, a considerable increase of the connective tissue of the liver was observed, and the connective tissue itself was studded with lymphoid bodies. In the earlier stage of the disease this appearance, however, is not always met with. The connective tissue may be increased in amount, but no lymphoid corpuscles seen among the fibres. When these are present they are most abundant in the sheath of the vessels; and it is around the interlobular vessels that the increase of the connective tissue is most marked. There can then be no doubt as to activity of the process going on in the connective tissue of the liver, but if these lymphoid bodies be absent it is a matter of some nicety, requiring considerable experience in the histology of the natural liver, to decide if an increase of the connective tissue have really taken place.

In the later stages the increase of the connective tissue becomes far more marked, and the lymphoid bodies are never absent throughout the whole of the liver. At least, I have always been able to see them in some part of the liver in all the cases of advanced nutmeg liver which I have examined. The connective tissue is strewn with them just as in primary cirrhosis. They are often very abundant indeed; but they do not show any linear arrangement as in cirrhosis, but are scattered irregularly through the tissue. This lack of arrangement may be due to the fact that but a small piece of the connective tissue is visible at one time under the microscope, so that it is well-nigh impossible to follow for any long distance the same tract of overgrown connective tissue.

The connective tissue is not always developed to the same

degree in every part of the same liver. Often quite close to spots where the interlobular tissue is actively engaged, crowded with lymphatic corpuscles, and much increased, a spot may be found quite free from these lymphatic bodies, and seemingly not greatly increased in bulk. This is an exceedingly common appearance in nutmeg liver, and one for which the observer must be prepared in forming a judgment upon it.

I have not found the connective tissue in any of the specimens which I have examined so greatly increased as to penetrate within the lobules themselves, although I can well conceive such an appearance in far advanced specimens of the disease to which the name of atrophous nutmeg liver has been given. I must still, however, be allowed to doubt, with Dr. Handfield Jones, if the growth of the interlobular tissue ever becomes so great as to give a hobnailed appearance to the liver.

It has been laid down as a pathological law by Sir William Jenner that, whenever an organ is mechanically congested for any length of time, the part becomes indurated, and also that an overgrowth of connective tissue takes place, although this is rather a matter of inference from Sir William's words than a direct statement[1] of his. He found in cases of congestion of the heart that the interstitial tissue lying between the muscular fibres was increased. It is also well known that one of the results of long-continued mechanical congestion of the kidney is an overgrowth of the intertubular connective tissue. It is not, therefore, surprising to find like changes in the interlobular tissue of the liver in mechanical congestion of that organ. The overgrowth cannot be due, as Rindfleisch implies,[2] to a mere coincidence, as I have found it in almost all the specimens which I have examined, even in the earliest stage of the

[1] William Jenner, 'Med.-Chir. Trans.,' 1860, vol. xliii, p. 200. The exact words are: "It is a pathological law that mechanically induced congestion, if long continued, slowly formed, and intermitting altogether or in degree, has for effect induration, and also *when the fibrin exuded is not of the powerfully contractile variety*, permanent increase in bulk."

[2] Rindfleisch, 'Lehrbuch der path. Gewebelehre,' Leipzig, 1867, p. 883.

disease; nor can it be merely an early stage of cirrhosis caused by habits of intemperance, for I have found the same interlobular overgrowth in four nutmeg livers of children varying from eight to twelve years in age, and who were unlikely to have been of intemperate habits. The presence of the lymphoid corpuscles I look upon only as evidence of the activity of the interlobular growth, and in no way related to a chronic inflammation.

The shrinking in the after stages of nutmeg liver is, in my opinion, due to the same cause as in cirrhosis, that is, to an overgrowth in the capsule of Glisson. A decrease in size cannot be caused by an atrophy of the parenchyma of the liver, so long as this atrophy is commensurate with the pressure upon the parenchyma by the dilating blood-vessels, for the decrease in size of the cells will be exactly counterbalanced by the increase in size of the blood-vessels. According to many authors, a new growth springs up in the midst of these dilated vessels, and it is by the growth of this new tissue that the decrease in size of the liver is brought about. I think this is but an imperfect explanation of the decrease in size, and it would seem more probable that if there be an interlobular growth, that the shrinking of the liver is due to the same cause as in cirrhosis.

In conclusion, I should like to add a few words as to the name of red atrophy which has been given to the atrophous nutmeg liver by Rindfleisch. Nothing can be more confusing than to have the same name given to two different states. The name of red atrophy was years ago given by Virchow[1] to a rare state of liver met with after wasting diseases, such as typhoid. This name has been accepted in this sense by pathologists such as Rokitansky[2] and Foerster,[3] and it is scarcely worth while to introduce a disturbing element into the nomenclature when the name of atrophous nutmeg liver is sufficiently good, and no advantage can be attained by change.

[1] Virchow, 'Arch. f. path. Anat.,' Bd. v, p. 289.

[2] Rokitansky, 'Lehrbuch d. path. Anat.,' 1861, iii, 244.

[3] Foerster, 'Handb. d. path. Anat.,' 1863, ii, 169.

ON THE

CONSTRUCTION AND USE

OF

A NEW FORM OF CARDIOGRAPH.

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(Received February 18th—Read May 11th, 1875.)

THE cardiograph, like the sphygmograph, has retained in the most essential particulars the construction first given to it by Marey, to whom the graphic method owes its chief development, and the alterations introduced by others are improvements of the mechanism in comparatively minor points. The chief feature of the instrument as invented by Marey is that, in order to facilitate the arrangement of the apparatus containing the clockwork, the motion to be recorded is transmitted through a flexible tube containing air. The cardiac motion of the chest-wall is imparted first to a cup or hollow disk, called a tambour or tympanum, the rim and back of which are rigid. The interior of the disk communicates by a tube of vulcanized india rubber, not too elastic, with a second tambour, in such a way that the two tambours and the tube enclose an air-tight cavity. The

front of the second tambour is covered by thin india rubber membrane, and the centre of this is attached to a light recording lever at a point not far from its axis of motion. By this means the motion communicated to the second tambour is magnified, and recorded upon a revolving cylinder called a polygraph. The amount of amplification given to the tracing can be varied at pleasure by adjusting with a screw the position of the recording lever.

Various forms of cardiograph differ from each other chiefly in the construction of the tambour which is applied to the chest over the apex of the heart. Marey first employed a metallic cup closed by a double membrane of india rubber, and having a tube by means of which water could be injected between the two layers of membrane. A convex elastic surface was thus obtained which, when applied to the chest-wall, slightly depressed the region of the heart. The contrivance, however, which he ultimately preferred is a slightly excavated cup, not covered by any membrane, but applied so closely to the skin that the contained air is hermetically enclosed. Within this cup is a spring ending in a pad of ivory, which slightly depresses the skin over the centre of impulse. In this way the motion of the whole surface of the chest contained within the circumference of the cup is communicated directly to the air within it, and thence to the column of air within the flexible tube.

In the use of this cardiograph a difficulty is found in keeping the cup closely enough applied to the skin, and both this and the former are open to the objection that the movement recorded is not that of any one point of the chest, but the mean result of the motion of a considerable surface. Both these disadvantages are avoided in the form of tambour introduced by Dr. Sanderson. The rim and back of this are made of brass, the front of elastic membrane. To the brass back is fixed a flat spring, which is bent twice at right angles in such a way that it overhangs the face of the tambour. Its extremity is perforated by a screw, the point of which rests on the centre of the mem-

brane, while its head is furnished with an ivory pad. The tambour has also three adjusting screws by which it rests on the wall of the chest. In this way the movement communicated to the elastic membrane and thence to the contained air is that of the very small area immediately in contact with the pad.

It is evident that if in any apparatus for the registration of motion anything of the nature of a spring intervenes between the moving object and the recording lever, the curve recorded will deviate from the true motion whenever any variation occurs in the tension of the spring, and any abrupt movement will be liable to set up oscillation in the instrument. The more flexible the spring the greater will be the risk of error. Now, in all the cardiographs hitherto described the recording lever is balanced upon a spring of air, which exceeds all other springs in sensitiveness, and therefore any sudden impulse communicated to the tambour is likely to set up oscillations whose period depends on the joint effect of the motion of the lever and that of the column of air. If, however, the movement to be recorded is sufficiently gradual and slow, it may be transmitted without any perceptible modification. This defect in the cardiograph has not escaped criticism by eminent physiologists, and it may be easily verified by trial that if any abrupt motion be imparted to the elastic membrane oscillations do take place. I cannot do better than quote the words of Professor Rutherford in his lectures on the circulation of the blood, published in the *Lancet* of November 25th, 1871. "You have seen," he says, "that if you merely touch for a moment the tambour of the cardiograph, you cause the lever of the polygraph in connection with it to describe, not a single wave, but a number of waves. There is first a great wave indicating the touch, and then a number of wavelets of diminishing size. These wavelets are due to the pendulum-like oscillations into which the column of air is thrown."

There is also another mode in which modification may be introduced into the curve. The motion of the heart is really transformed into waves of condensation and rarefaction

in the elastic fluid, and these waves are liable to a change in shape as they pass along the flexible tube. It may easily be shown by taking tracings at different points of the arteries of the body, or of elastic tubes containing water, which form a part of a schema representing the circulation, how considerable is the alteration in the form of the pulse wave as it proceeds. In the case of blood or water there is not only a gradual rounding of the curve, but other changes take place from the effects of the momentum of the fluid. For the purpose of the cardiograph, therefore, air is far more suitable than water would be, but the rounding off of sharp points or abrupt motions will occur much more rapidly in a tube containing air than in one containing water, for in the latter case this effect is produced only by the elasticity of the tube, but in the former it depends chiefly on the elasticity of the contained fluid.

In the case of the cardiograph this rounding off of any abrupt features of the curve is probably a positive advantage, for it counteracts, to a great extent, the effect of the other source of error, and the more gradual motion is less likely to call out the oscillations which would be set up if the movements communicated to the lever were as abrupt as the actual movements of the chest-wall.

For the verification of the results obtained by his cardiograph Marey has employed the method of imparting to the apparatus a motion already known, which can be compared with the curve described by the recording lever. For this purpose he made use of an eccentric turned by a handle. The circumference of the eccentric acted directly upon the short arm of a recording lever which was kept in close contact with it by a strong antagonistic spring. The eccentric was cut into such a shape that the curve described by this first lever resembled a cardiographic tracing as obtained by the polygraph. The lever was then attached to the membrane of the first tambour of a polygraph. The motion was thus transmitted by the tube to the second tambour and recording lever, by which a second curve was described upon the revolving cylinder immediately above the

first. If, then, the instrument were perfect in its indications, the two curves ought to correspond precisely. It was found that the slower the motion of the eccentric the more perfect was the identity between the two, but that if the eccentric were turned more rapidly the effects of the inertia of the instrument became manifest in the second curve. The discrepancy, however, could be made to disappear by increasing the friction of the pen against the paper.

The method of verification thus adopted by Marey is a most valuable and trustworthy one for indicating the limits of possible error. But as applied by him in this instance it proves at most that the cardiac motion, as transformed by a first transmission through the tube, and in which the main ascent and descent are probably already followed by one or more oscillatory repetitions, can be transmitted a second time without any further obvious change. It does not prove that it is possible so to transmit without modification the original motion of the chest-wall, which is shown by other means to have more suddenness than appears in the polygraphic curve. It is evident, indeed, that no very sudden motion could be communicated to the lever by the revolution of an eccentric having a curved margin with which its shorter arm is kept in close contact, even when the handle of the eccentric is turned rapidly.

The sources of possible inaccuracy in Marey's polygraph do not in the slightest degree invalidate the most important result which he obtained by its means, namely, the demonstration of the normal succession of events in the several cavities of the heart and in the aorta. This he obtained by inserting sounds, whose open extremity was covered by an elastic membrane, into the right auricle and also into the right ventricle of a horse, while another elastic ampulla was inserted in the chest-wall over the centre of cardiac impulse, and simultaneous tracings were procured of the three motions. In another experiment one sound was inserted into the aorta and another into the left ventricle. In none of these curves can any suspicion

attach to the chief points essential for comparison, namely, the main upstroke and main downstroke which denote the beginning and the end of the ventricular systole, and the slight elevation just preceding the ventricular upstroke which indicates the systole of the auricle. Some minor points, however, are seen in the curve which are probably the result of oscillation in the polygraph. This is especially the case with a descending series of waves superposed on the broad systolic summit, immediately after the principal upstroke, which are attributed by Marey himself to an oscillation in the tension of the auriculo-ventricular valves.

The sphygmograph itself, applied directly over the apex of the heart, has been employed both by Marey and also by Professor A. Garrod and other observers for the obtaining of cardiac tracings. The curves obtained in this way, especially those published by Professor Garrod, are much more complex than those described by the polygraph, and show a number of minor points, which appear to be lost when the motion is transmitted through a tube. In attempting, however, to make use of the sphygmograph in this way in cases of heart disease two insuperable difficulties are found. The first is that, when the apex beat is powerful and extends over a wide area, the ivory bars of the instrument touch the chest within the range of the heart's impulse. The curve described represents, therefore, not the motion of any part of the chest, but only the excess of the motion of the spring pad over that of the ivory bars. The result is that some of the features in the curve may often become reversed, and in some cases even the principal upstroke caused by the ventricular systole may be converted into a downstroke.

The second difficulty arises from the great variation in the extent of the cardiac motion. In the case of a healthy heart the ordinary sphygmograph, which magnifies the motion about ninety times in a vertical direction, may give a curve of moderate amplitude, but when there is much hypertrophy the motion is far too great to be recorded by such an instrument. Even with a magnifying power of

only twenty the movements of the lever may be so extensive that not only is the curve likely to be modified by the effects of its acquired velocity, but the writing point cannot be retained within the limits of the smoked paper.

The cardiograph represented in the engraving (see p. 360) has been constructed, according to my directions, with the view of obviating these two defects and obtaining the cardiac tracings in all cases directly from the chest. The brass frame of the instrument resembles that of the sphygmograph, except as regards the bar which carries the knife edge, A, through which the motion is transmitted to the long lever. This bar, B, is made up of two parts, of which one slides within the other, and can be fixed by means of a screw, C, in whatever position is desired. There is also a second knife edge, D, which can be raised or lowered at pleasure, attached to the same bar at a greater distance from the axis of the long lever. By this means the magnifying power of the instrument, as regards the vertical height of the curve described, can be varied from ten to about a hundred. The brass frame, which in the sphygmograph is rigidly fixed to two parallel bars of ivory by which it is supported, is freely suspended in the cardiograph by means of two transverse rods of steel, E. These are attached by joints, F, which allow both of vertical and horizontal adjustment, to four vertical rods of steel, G, each pair of which is inserted into a bar of wood covered with leather,—by means of these wooden bars the instrument rests upon the chest. They can be separated to a width of nearly five inches, and the instrument can be raised or lowered at pleasure at either end, and in this way it can be adapted to a chest of any size or shape.

In order that vertical adjustments at either end may be possible independently, the brass frame is not in immediate contact with both the transverse bars which support it, but at one end it is suspended by an intermediate piece of brass, I, which, when the instrument is in position, is tightened and made rigid by a screw. The spring
is employed to press upon the centre of impulse is

FIG. 1.

in a mode similar to that adopted in the sphygmograph. The mechanism, however, by which the amount of pressure is finally adjusted is a simpler one than that employed in any one of the various forms of sphygmograph now generally used. This simplification is rendered possible by the fact that, in the case of cardiographic tracings, a knowledge of the exact amount of pressure employed would have little or no significance. The adjustment is effected by means of a screw, K, which perforates the short arm of the spring lever, B. The weight of the lever itself is also counterbalanced by a small antagonistic spring.

In this way the pressure upon the point at which the spring pad is applied can be reduced almost to zero, and thus it is easy to obtain with this instrument a tracing representing the backstroke in veins (Fig. 3), which even the weight of the spring of an ordinary sphygmograph is generally sufficient to extinguish.

There are also two small springs, L, of different strength, to depress the long lever and prevent its being jerked away by any sudden motion from the knife edge on which it rests. Either of these can be used or turned aside at pleasure. When the instrument is used in a vertical position it is generally better to dispense with this small spring, since it adds a little to the friction, and it is found that the lever does not become separated from the knife edge, provided that the magnifying power of the cardiograph be so adjusted that its movements have only a very moderate amplitude. If, however, it is desired to take a tracing from a patient in a sitting or standing posture, it is necessary to use the secondary spring, for then the recording lever is no longer kept in position by its own gravity.

The cardiograph may be fixed upon the chest by two narrow straps passed round the body and fastened by buckles. These should be partly elastic, that they may yield a little if the patient makes an inspiratory effort while the clockwork is in motion. In this way the disturbing influence of the muscular movement upon the

cardiac curve is diminished. As soon, however, as the observer has acquired some dexterity, it will be found sufficient in most instances to hold the instrument against the chest with the hand. In the case of a healthy heart, a uniform and perfect tracing can, as a general rule, be obtained only while the breath is held, and it is best that it should be stopped at the end of expiration. When, however, there is much hypertrophy, the magnifying power required is so much less that the respiratory curve becomes little noticeable in proportion to the cardiac motion, and then the holding of the breath is not so essential. In many cases, when the impulse is very feeble, a more ample curve may be obtained if the patient is placed in a sitting posture, leaning somewhat forward.

In the tracings taken by the direct cardiograph the motions are much more abrupt than in the curves obtained with the ordinary instrument, and the minor elements in the trace are more numerous. This result tends to confirm the conclusion drawn from other grounds, that the transmission through the tube containing air introduces some modification into the motion. With this instrument, moreover, there is greater facility for ascertaining the relation of the chief elevations or depressions of the curve to the impulse or thrill felt by the finger and the sounds or murmurs heard by the stethoscope. For with the tambour cardiograph a slight interval of time is required for the waves to pass along the intervening tube, but in this the movements of the lever follow immediately those of the chest-wall.

It is easy, therefore, for the observer to watch the lever while he rests a finger upon the apex or listens to the heart sounds with a flexible stethoscope, and in this way he may ascertain the significance of the chief features in the curve. Not unfrequently it is found that the thrill which accompanies a murmur is represented in the trace by a vibratile line, and when this is the case a permanent record is obtained of the precise relation of the murmur with respect to the systole and diastole (see Figs. 6 and 11).

With regard to the possibility that the curve may be modified by the effect of acquired velocity in the instrument, the direct cardiograph, like all other recording instruments, fails to secure absolute fidelity. For the elastic tissues which intervene between the pad of the lever and the heart are in some degree analogous to a spring, and by a variation in the degree of their compression a deviation in the curve might arise. Such variation is likely to be greater, the smaller the pressure employed.

In the case of the heart, since the moving force is greater than that of the radial pulse, a much greater pressure may usually be applied, and, therefore, the liability to oscillation is less than with the sphygmograph, and, *à fortiori*, less than in the tambour cardiograph, in which the lever rests upon a spring of air. As compared with the sphygmograph also, since the amplification in vertical height given to the motion is generally many times less, so many times less also is the effect upon the trace of any given error in the instrument. Another source of error might arise from the long lever becoming separated, in consequence of its acquired velocity, from the knife edge by which it is moved. This is especially likely to occur when the heart's impulse is sudden and strong, and it is a more probable occurrence in the case of the cardiograph than with the sphygmograph, since the motion to be recorded is often much more abrupt. It may be avoided by the use of the small secondary spring, L, which is used to depress the long lever; but in most cases this may be done as effectually, and with less disadvantage, by so adjusting the magnifying power of the cardiograph that the movements of the lever are not too extensive. As a rule, however, it is desirable to compare tracings taken with and without the secondary spring. Since the main upstroke in the curve is usually very sudden, it is probable that the summit which forms the commencement of the systolic eminence often owes something of its height and sharpness to the effect of acquired velocity, but it would seem that, if due precautions be

taken, the rest of the curve may be relied upon as faithful to the truth.

It does not come within the scope of the present paper to discuss the difficult question of the interpretation of all the elevations seen in the cardiographic tracings, a problem which I have attempted to solve in an article contributed to the 'Guy's Hospital Reports' for 1875, but it may be well briefly to mention some of the most important characters, as seen in the specimens shown in the woodcuts. The systolic part of the trace forms a somewhat square eminence (see Figs. 2, 4, and 10), of which the most important elements are two—first, a sharp-pointed summit at its commencement, probably due to the hardening and rounding of the ventricles; and secondly, a more rounded swelling towards its conclusion, probably caused by the continued pressure against the ribs as the heart is propelled forwards. The former of these is increased in proportion when the heart is dilated, and the latter when it is hypertrophied, and, therefore, by a comparison of the two some estimate may be formed of the ratio which hypertrophy bears to dilatation in any given case.

The diastolic part of the curve, when the heart is healthy, forms a very gradual ascent, commencing at the end of the principal downstroke (Fig. 2). In cases of aortic incompetence a rapid rise takes place, the degree of which indicates the freedom of the regurgitation (Fig. 4). In mitral regurgitation the ascent is also somewhat quicker than normal, in consequence of the rapid repletion of the ventricles caused by high tension in pulmonary and systemic veins.

An important application of the cardiograph is found in the indications which it affords in cases of mitral contraction. In the typical healthy curve (Fig. 2) the auricular systole is expressed by a small elevation just preceding the main upstroke. In some cases of mitral contraction in which a short presystolic murmur is heard, the auricular elevation is increased in height, and is either normal in width or slightly prolonged (Fig. 5); in others, in which a

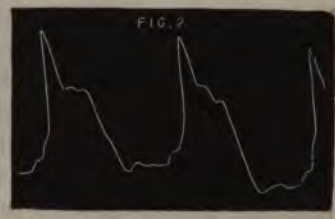

FIG. 2.—Normal apex tracing.

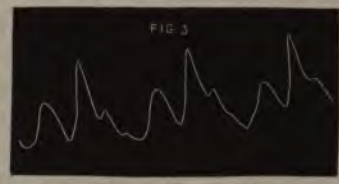

FIG. 3.—Tracing from the subclavian vein in a case of tricuspid regurgitation with mitral contraction.

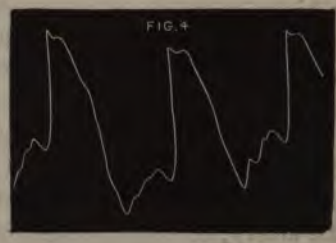

FIG. 4.—Apex tracing. Free aortic regurgitation.

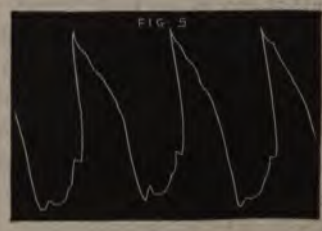

FIG. 5.—Apex tracing from a case of mitral contraction in which a short presystolic bruit was heard.

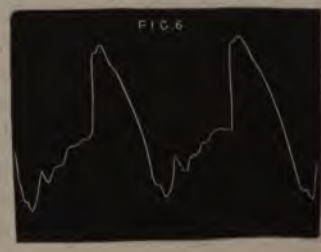

FIG. 6.—Apex tracing. Long and harsh presystolic murmur, accompanied by thrill, running up to the first sound.

harsh and characteristic presystolic murmur extends over the whole period of rest, it appears to be replaced by a vibratile line which begins to ascend almost from the commencement of the ventricular diastole (Fig. 6). But in another class of cases of mitral contraction, in which there is a soft, or even sometimes a purring or somewhat rumbling murmur, commencing with the ventricular diastole, and either reaching up to the succeeding systole or separated from it by an interval, the character of the curve is altogether different, and appears to point to a different causation for the bruit (Fig. 7). The auricular elevation is either altogether invisible or it appears in its normal position and not prolonged.

There is another important use of the cardiograph in which it serves, not to afford evidence on a theoretical question of the mechanism of a murmur, but as a practical aid to diagnosis. This is found in its application to any point of abnormal pulsation either in the chest or abdomen. In the chest it may be necessary to distinguish between impulse communicated from any one of the cavities of the heart, the pulsation of a tumour, the simple dilatation of an artery, or actual aneurism. The form of the curve in all these cases is different, and in the case of an aneurism some information is afforded by its appearance as to the freedom of communication between the sac and the artery. The ordinary sphygmograph is not usually available for this purpose, since the pulsation is generally too forcible and too diffused to allow a reliable tracing to be obtained by its means. The tracing shown in Fig. 8 was taken from the surface of an aneurism of the arch of the aorta. It will be seen that it is comparable with the sphygmographic tracing of an arterial pulse. The so-called percussion wave is just visible in the ascent, and the dicrotic wave is seen in the descent. When there is no aneurismal sac, but only a dilatation of the aorta, the correspondence is much closer, and the tracing may exactly resemble that of a senile pulse obtained from the radial artery. This result is of interest as showing the fallacy of the view which has been held by

FIG. 7.—Apex tracing from a case of mitral contraction in which a blowing diastolic murmur, followed by a pause, was heard at the apex only. There was also a short systolic murmur, and a presystolic murmur had been heard previously.

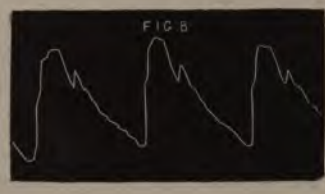

FIG. 8.—From the surface of an aneurism of the arch of the aorta, which formed a pulsating swelling displacing the sternum.

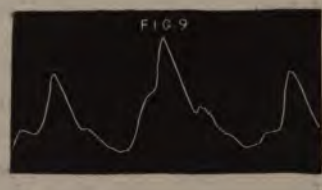

FIG. 9.—From the surface of the liver in a case of tricuspid regurgitation with mitral contraction, taken during respiration.

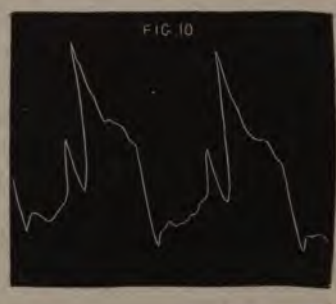

FIG. 10.—Apex tracing from a case of old mitral disease in which a systolic murmur was heard. After death the mitral orifice was found widely dilated, the left auricle much hypertrophied.

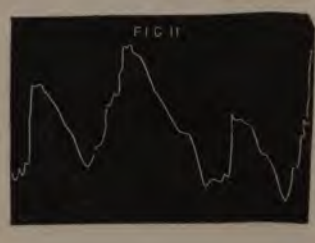

FIG. 11.—Apex tracing from a case of pericarditis in which there was a to-and-fro friction sound accompanied by thrill.

some, that the dicrotism of the pulse is a phenomenon which occurs only in the peripheral arteries, and not in the aorta itself.

In the case of abdominal pulsation the diagnosis may lie between cardiac impulse communicated through the liver, expansive pulsation of the liver due to backstroke in the hepatic veins, temporary dilatation of the abdominal aorta produced by nervous influence, the transmission of pulsation by a tumour, and aneurism. In this case also the transmitted cardiac impulse or the aneurismal pulsation may be distinguished by the shape of the curve. The tracing in Fig. 9 was taken from the surface of the liver in a case of mitral contraction with tricuspid regurgitation, in which there was marked pulsation also in the external jugular veins. It shows the typical features of a venous pulse, namely, the anadicrotic wave, preceding the principal summit and caused by the auricular systole, and the katadicrotic wave, which breaks the descent and appears to be due to an oscillation analogous to that which produces the dicrotic wave in the arterial pulse. The last tracing should be compared with that shown in Fig. 3, which was taken with the cardiograph from the subclavian vein of another patient in whom there was tricuspid regurgitation and mitral contraction, the auricular elevation being very strongly marked in the apex tracing. In this case the wave which is due to the auricular systole, and precedes the principal eminence, has a very great proportionate magnitude and distinctness, and this curve may be commended to the attention of those who find it difficult to believe that the auricle can, in any case, acquire force sufficient to produce a murmur by its contraction. In the descending curve are two secondary waves which seem to correspond to the dicrotic wave, and the tricrotic wave or second oscillation which is sometimes also seen in the arterial pulse tracing when tension is low.

ON A CASE

OF

DOUBLE FACIAL PALSY,

WITH OBSERVATIONS ON THE

PHYSIOLOGY OF THE NERVES SUPPLYING THE FORE PART OF THE TONGUE.

BY

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In the fifty-second volume of the 'Transactions' of this Society a case is recorded by Julius Althaus, M.D., of complete loss of function of the whole fifth pair of cerebral nerves unaccompanied by any other affection of cerebral or nervous matter. The case, as the author of it observed, afforded an excellent opportunity of thoroughly investigating the functions of the trifacial nerve with special regard to certain points still involved in obscurity. The case which I have now to record is an analogous case of complete loss of function of the portio dura of the seventh pair of nerves, also unaccompanied by any other affection of cerebral or nervous matter. These two cases may be regarded as completely supplementary to each other, and appear to me when

taken together to throw light especially on the functions of the chorda tympani.

Joseph D—, an intelligent young man, 24 years of age, was admitted to Dr. Steevens' Hospital, Dublin, on September 23rd, 1873. The first thing that attracted one's attention about him was that in speaking he could not pronounce the labial letters. On further examination it became clear that he was affected with paralysis of the portio dura of the seventh pair on both sides of the face.

History—He is by occupation a fireman in the employment of the Great Southern and Western Railway Company of Ireland; quite steady in his habits of life, and without previous syphilitic infection. Up to three weeks ago he was in the enjoyment of good health; the left side of the face then became affected, and one week later the right. The patient himself attributes his attack to wet and cold aggravated by his having been, at a moment when the weather was unusually cold for the season, transferred from an engine of the slow "goods train" to the much faster mail and express train.

His expression is remarkable, or rather his absolute want of expression. All the muscles of expression are palsied; he cannot smile or frown, blow, whistle, or raise his brows; he cannot shut his eyes or close his lips. All the symptoms of palsy of the portio dura of the seventh pair are equally well marked upon each side.

A careful examination was made of the condition of all the other nerves of the head, as it was feared, on account of its double character, that the facial palsy might have a central origin. The following was the result:

1st pair.—The sense of smell is quite normal.

2nd pair.—There is no disturbance of vision.

3rd pair.—There is no squint, and all the movements of the eyeball are perfect.

4th pair.—There is no evidence of any affection of the superior oblique muscle.

5th pair.—Feeling is normal everywhere over the skin of

the face; in the tongue, touch and thermic impressions are perfectly perceived, *but he cannot taste on the forepart of the tongue.* The muscles supplied by the motor branches of the fifth appear unaffected. The patient can crack a nut between the molar teeth on either side with ease. There is no sign of palsy of the soft palate; the movement of its muscles in attempting to produce guttural sounds when the mouth is wide open is strong and symmetrical.

6th pair.—The external rectus is absolutely unaffected.

7th pair.—The portio dura paralysed on each side, and none of the muscles of expression respond to even strong Faradisation; the sense of hearing quite normal and equally good on each side.

8th pair.—The patient can taste on the base and back part of the tongue through the part supplied by the glosso-pharyngeal nerve.

9th pair.—All the motions of the tongue are perfect; there is no difficulty in swallowing save that the lips cannot be closed in drinking, and he has to throw back his head like a hen when drinking.

The muscles of the face do not contract under the influence of Faradisation; the effect of an interrupted voltaic current, however, is different.

October 30th.—On this day it was first observed that the corrugator supercilii muscles of the left side contracted during Faradisation, and slight twitching took place among the fibres of the orbicularis palpebrarum on the same side. On the right side of the face there is still not one single muscle which responds to Faradisation. There is a slight reappearance of voluntary motion in the left corrugator supercilii; a slight but distinct contraction is to be observed when the patient endeavours to frown.

November 17th.—The patient left hospital to-day, but continues to attend twice a week as an out-patient. The muscles of the brow, corner of the mouth, and cheek are slowly but decidedly gaining power on the left side. There is as yet no sign of motor power returning on the right side in any one muscle.

January 1st, 1874.—Patient much improved on the left side of the face; right side also regaining motor power in the muscles of the mouth, but not as yet over those of the eyebrow and eyelid; the sense of taste still, as before, absent from the forepart of the tongue; general health and appearance good.

April 18th.—The patient was examined this day. The facial muscles may be now said to have regained their powers. He can shut both eyes, pronounce the labial letters, close his mouth, whistle, smile, frown, &c. The movements on the right side are not yet quite so perfect as on the left. The corrugator supercilii especially is more sluggish than on the left side. The sense of taste although returning in some degree, is still deficient in the forepart of the tongue.

On the 23rd December, 1874, I last saw this patient. The left side had recovered more perfectly than the right, although he has recovered power of expression, with a slight contraction observable upon the muscles of the right side, which are sluggish in their movements. A casual observer, however, would not now notice any traces of facial paralysis. The sense of taste in the forepart of the tongue is still obscure, and the secretion of saliva scanty on the application of sapid bodies to the forepart of the tongue.

It seems, therefore, that this case was one of double paralysis of the portio dura of the seventh pair accompanied with loss of the sense of taste in the anterior portion of the tongue. There is evidence, as clear and precise as clinical evidence can be, that no other nerves were engaged save those presiding over the motions of the facial muscles and the sense of taste in the anterior part of the tongue. The case being one the symptoms of which were remarkably neatly and well defined, and the patient himself very intelligent, it was possible to investigate some details with scrupulous care. The case was, in fact, free from many of the disturbing causes which tend to obscure observation and baffle all attempts to get positive and reliable facts.

Regarding the case as one in which there was no central lesion, but an uncomplicated palsy of the portio dura of the seventh pair, engaging also the chorda tympani, the following comparative observations were made with the view of determining the functions of the latter nerve:

His eyes being shut, the patient could detect the slightest touch on the forepart of the tongue. He could perceive a light touch with a feather, a camel's hair brush, or even a single hair, as well as I or any of the students present. He could with the tip of the tongue distinguish the double points of the æsthesiometer as well as any of us.

Two egg-cups being filled with water slightly differing in temperature, and, the eyes being closed, the tongue applied first to the one and then to the other, the patient could perceive without hesitation or mistake a difference not amounting to more than four or five degrees. Compared with myself and the students present the power of appreciating differences of temperature appeared normal.

The sense of taste was completely lost on the forepart of the tongue. The patient could not detect any difference between solutions of sugar, citric acid, salt, and quinine. Comparative trials were made in this way:—Solutions of the above substances were made in mucilage of gum, so that all were of uniform consistence. The words "sweet, sour, salt, bitter," and "cannot tell" were written on a slip of paper, and the patient directed, after the solution had been applied on the edges and forepart of the protruded tongue with a camel's hair brush, to reply by pointing to the answer without withdrawing the tongue into the mouth. His unfailing reply was to point to "cannot tell," while the rest of us, although occasionally requiring a moment or two, always answered correctly.

The patient could not perceive the electrical taste when the poles of a battery were applied on the forepart of the tongue.

The secretion of saliva excited by the application of sapid substances to the forepart of the tongue was in abeyance. The comparative observations made on this point were very

striking :—The patient and one of the students were placed sitting with their mouths open and their tongues raised towards the roof of the mouth, so that one could see in beneath the tongue within the incisor teeth; a camel's hair brush dipped in lemon juice was then passed over the tip and edges of the tongue; in the healthy individual, in less than half a minute, the hollow beneath the tongue became filled with saliva, which dribbled over the lip, while in the patient no saliva could be seen welling up.

The temperature within the mouth was normal.

In recording this case I wish to confine myself as closely as possible to the accurately observed symptoms. The chief interest of it lies in its comparison with Dr. Althaus's case already referred to. In his case ordinary sensation was lost in the tongue, while the sense of taste remained. He thus describes it :—"The mucous membrane of the mouth was completely anæsthetic as far as the gums, the tongue, the inner surface of the cheeks, and the hard palate were concerned. The tongue presented a most frightful spectacle, having been severely bitten and lacerated in every direction during the act of taking food, the patient being entirely unconscious of his biting the tongue whenever he did so. Some parts of the inner surface of the cheeks had also been bitten and were badly ulcerated. The sense of taste was not lost, for the patient tasted salt, sugar, and quinine on the front as well as on the back part of the tongue. Yet in the anterior part of that organ the perception of taste seemed to have lost its quickness, for while on my applying sapient substances to the posterior part of it the patient would at once exclaim 'bitter,' 'sweet,' 'salt,' he took about five or six seconds on the front part of the tongue to describe the taste, but he never made a single mistake there. He also perceived the galvanic taste of five cells of Daniell's battery, if applied directly to the tongue." In the case recorded by me ordinary sensation was unimpaired, but the sense of taste was lost. The two cases taken together go clearly to show that the so-called gustatory nerve is in reality a compound nerve, endowed with the sense of taste through the

fibres coming to it through the chorda tympani, while the ordinary sense of touch belongs really to the fifth pair. Upon the vexed question of the precise channel through which the taste-fibres enter the cranium, whether through the fifth, seventh, or glosso-pharyngeal, the case obviously throws no light. It will require further observations to clear up this obscure part of the subject.

ON THE SO-CALLED

PARTIAL DISLOCATION OF THE HUMERUS.

BY

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COMMUNICATED BY

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Received April 13th—Read May 25th, 1875.

Even at the present day opinion is divided as to whether a partial dislocation of the humerus should be included in the classical description of dislocation of that bone or not For my own part, I cannot understand the nature of an injury which would be sufficient to force the head of the bone through the upper part of the capsular ligament without causing fracture of one or both of the overlying scapular processes.

Examples of dislocations of the humerus *with* fracture of the coracoid processes have been recorded in the 'Transactions' of this Society by Mr. Holmes and Mr. Prescott Hewett. Such injuries, however, would not be included in the generally accepted definition of the "partial dislocation."

But even supposing that an injury had forced the head of the humerus into immediate contact with the under surface

of the acromion process, why, we may ask, should it remain there? It cannot be from the bone being grasped below the equator of the head by the ruptured capsular ligament, for there is not sufficient room between the capsule and the process for the head to have passed far enough out of the capsule for such a condition to have resulted.

In the two well-known cases to which I am about to refer, and upon which the usual description of the partial dislocation has been founded, the capsule and the surrounding muscles were described as being entire, or else the ligament was "slightly ruptured." There has never been, so far as I can ascertain, any unequivocal instance of a subluxation of the humerus, and those authors who describe such a lesion base their account of it either upon the description given by Sir Astley Cooper in his grand work upon fractures and dislocations of the joints, or else upon the account of a peculiar condition of a shoulder-joint given by Mr. Soden in the 'Transactions' of this Society for the year 1841. It is my endeavour in this paper to show that the changes noticed in these preparations were the result of disease and not of accident. And in support of this I shall describe the right and left shoulder-joints of two very old female subjects which were examined by me in the dissecting room of St. Mary's Hospital during the last two winter sessions. The right joints bore a very close resemblance to that described by Sir Astley Cooper, whilst both the left joints corresponded very closely with that which came under the notice of Mr. Soden. It must be admitted that it is hardly likely that both these old women could have been subjects of partial dislocations of both humeri, the result of accidental violence.

Before going further, however, I would like to point out briefly the views held by various authors on the subject of the partial dislocation. Amongst those who admit it are—

Sir William Fergusson, who instances Soden's case, and who also describes specimens which he had examined in the dissecting room; of the latter, however, Sir William says, "Whether these effects were the result of violence or of gradual change, I cannot say."

Erichsen, who gives Soden's case, and who believes that the subluxation can only take place when the tendon of the biceps is displaced from its groove or ruptured.

James Lane, in Cooper's 'Dictionary of Practical Surgery,' who quotes Soden's case, and one which was described by Mr. Alfred Smee, and to which I shall refer further on; and

Pirrie, who quotes Sir Astley Cooper's case, and another dissecting-room specimen.

On the other hand, *Hamilton* believes that the anatomical structure of the joint renders the occurrence of such an injury exceedingly improbable, if not impossible, whilst

Flower and *Hulke*, in Holmes's 'System of Surgery,' deny that the partial dislocation as the result of accident has ever been satisfactorily proved, either in the living or dead subject.

Robert Adams, in 'Todd's Cyclopædia,' attributes the lesion described as subluxation by various authors, to chronic disease or congenital malformation.

Then, having given an account of my own preparations, I will place their analogues in close connection.

Case 1.—On cutting across the right deltoid of the first subject an enormous bursa was discovered in communication with the articular synovial membrane through a well-defined elliptical opening in the upper part of the capsule. Through this aperture the head of the humerus was enabled to articulate with the under surface of the acromion process, where a smooth articular facet had become developed. The muscles surrounding the joint were entire. The capsule was attached to the coracoid and acromion processes, and there was a considerable development of cartilage and of vascular fringes in different parts of the joint. The circumference of the head of the humerus was increased in size by the growth of tuberculated pieces of bone and cartilage; and as the articular cartilage was thickened in some places, and eroded in others, the globular mass of bone was of a very irregular shape. The intra-articular portion of the biceps was ruptured, and the lower part of the tendon was displaced from the bicipital groove and adhered firmly to the lesser tuberosity.

Case 2.—The right joint of the second subject was very similar to that just described. The subdeltoid bursa was very large, and the tendon of the biceps was ruptured, and was adherent to the upper part of the bicipital groove. The head of the humerus was much enlarged by osseo-cartilaginous deposit, and was pressing up underneath the acromion process and the coraco-acromial ligament, and there was a corresponding erosion of the articular cartilage. The two surfaces of bone, however, were not in immediate contact, as the humerus had not sufficiently worked its way through the loose capsular ligament when death had occurred.

Here follows the account of Sir Astley Cooper's specimen, which was obtained from the dissecting room of St. Thomas's Hospital in the year 1819. The account is transcribed from Sir Astley's work almost literally:—"The head of the humerus was placed more forward than is natural. The tendons of those muscles which are connected with the joint were not torn, and the capsular ligament was found attached to the coracoid process of the scapula. The head of the os humeri was situated under the coracoid process which formed the upper part of the new glenoid cavity. The natural rounded form of the head of the bone was much altered, it having become irregularly oviform with its long axis from above downwards. There was considerable deposit of cartilage in the rest of the glenoid cavity and upon the head of the humerus. The long head of the biceps muscle seemed to have been ruptured near to its origin (at the upper part of the glenoid cavity), for at this part the tendon was very small, and had the appearance of being a new formation." (The rest of the short description is of no material importance; the right joint does not seem to have been examined.)

These three descriptions agree in their most important features: viz., in the alteration in shape and position of the head of the humerus (the enlarged head lying against one of the scapular processes), in the rupture of the biceps tendon, in the new growths in the joint, and in the uninjured condition of the small rotator muscles.

The left joints of my two subjects presented many features corresponding to those of the right side, but the disease was in all respects less advanced, so that the tendon of the biceps, though worn very fibrous and ragged, had not yet given way. In subject No. 2 it was very thin and frayed out. In both cases, however, it had been displaced from the bicipital groove by a new osseous growth, and was dislocated on to the lesser tuberosity of the humerus, where it played in a separate sheath of fibrous tissue and synovial membrane. These specimens closely resemble that described by Mr. Soden, of which a short account is here subjoined.

In May, 1839, a rheumatic man, thirty-nine years of age, fell on to his right elbow whilst rising from nailing down a carpet. Great pain then came on in the corresponding shoulder, but he could raise his arm over his head, and went on working till the pain compelled him to desist. Next morning the joint was greatly swollen, but Mr. Soden satisfied himself that there was neither fracture nor dislocation. Three weeks afterwards, when the swelling had disappeared, Mr. Soden gave the joint another examination, when he experienced a crepitating sensation upon moving the limb. He also found that the arm could be but little raised, on account of the great tuberosity coming into contact with the acromion. There was also undue prominence in front.

Six months afterwards the man died in the Bath Hospital of fracture of the base of the skull, and a subsequent examination of the joint gave the following results:—The capsule, but slightly ruptured, was thickened and adherent. The synovial membrane was vascular and inflamed. The head of the humerus was in contact with the acromion process, and its articular cartilage was ulcerated at that spot. The tendon of the long head of the biceps was entire, and, dislocated from the bicipital groove, lay in a synovial sheath upon the lesser tuberosity of the humerus. The preparation is now in the museum of King's College, where I have examined it, as far as I am able. The head of the bone, as in the other cases, is considerably enlarged, and its circumference rendered irregular by new bony or cartilaginous

growths. In the adjoining bottle in the museum (1341 (4)) is a preparation from Sir William Fergusson's collection. It is described in the catalogue as "an old dislocation;" and as the articular vessels are filled with injection, I infer that it is one of those dissecting-room specimens of whose nature the great surgeon gave so cautious an opinion. It seems to be in every respect similar to Sir Astley Cooper's preparation, and to the two right joints which I have just described. It is not a little strange that they should, all four, be dissecting-room specimens.

Soden's preparation, then, and my two left joints, agree in almost every respect. They differ from Cooper's and from my right joints only in this, that the disease is less advanced. It is not unlikely that the right joint is more prone to disease than the left. In a little time the tendon would, in my cases at least, have worn through. As it was, it was merely dislocated, as described by Soden, and the cause of this dislocation I attribute to a gradual filling up of the bicipital groove.

A most important point in connection with my second pair of shoulder-joints is, that the knee-joints of the same subject were also affected with a chronic arthritis. The patella shows considerable bony deposit, whilst its posterior surface and the trochlear aspect of the femur are in places entirely denuded of cartilage, and are worn by friction in longitudinal streaks. [These parts of the left side were exhibited.]

Mr. Callaway gives the account of twenty-two (22) preparations of the shoulder-joint in which there has been found either a dislocation or a rupture of the tendon of origin of the biceps, and states his belief that the partial dislocation of the humerus is due to one of these peculiar conditions.

He includes in this series the preparations of Sir Astley Cooper and of Mr. Soden, and also some which Robert Knox had described in the 'London Medical Mirror,' which were in every way similar to those now before the Society. He thought that such lesions were rare, or that they were rarely

reported. They were, I apprehend, all dissecting-room specimens.

Shortly after, and in the same publication ('London Medical Mirror,' 1828), Mr. Stanley gave an account of three similar specimens. Callaway includes these also, and a dissecting-room preparation which was described in the 'Lancet' for 1845 by Mr. Alfred Smee, of the Aldersgate School of Medicine. This last joint is of further interest, for the disease in it was so far advanced that not only was the tendon of the biceps ruptured and adherent to the head of the humerus, but the greater tuberosity was almost all articular, and was of necessity deprived of its muscular connections. Mr. Smee could hear of no case like his but that of Mr. Soden; but this, he went on to say, differed from his own, in that the tendon of the biceps was merely dislocated. With the exception of Mr. Soden's case, however, he considered that his own was without precedent.

The great similarity existing between my own shoulder-joint preparations (which are clearly affected by a chronic disease, probably of rheumatic origin) and those to which I have referred, leads me to this conclusion, that the specimens described by Cooper and Soden were examples of the same pathological change, and cannot, therefore, be taken as instances of "partial dislocation" in the proper sense of the term. And I am of opinion that, until some unequivocal example of the partial dislocation be adduced, the description of the lesion as the result of injury should be omitted from our text-books, more especially since the anatomical structure of the joint contra-indicates the possibility of its occurrence.

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